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ESTROGENS AND THEIR EFFECT ON CILIATED MUCOSA

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The nature of a relationship between the nasopharyngeal mucosa and the gonads is a question which has attracted the attention of investigators now and then from the time of Hippocrates to the present.[1a] Apart from some observations on the connection between epistaxis and cyclic activity of the uterine mucosa, the problem had little practical interest for members of the medical profession until Mortimer, Wright and Collip [1b] reported observations which linked atrophic rhinitis and ozena to a pituitary dystrophy and encouraging results from the treatment of patients with such conditions by nasal insufflation of estrogenic material. Later, further encouraging results were reported from the Montreal clinic [1c] on the use of this type of therapy in the treatment of patients for "constitutional deafness."

Confirmation of the beneficial effects of estrogenic topical therapy in cases of atrophic rhinitis was obtained by Blaisdell [2] and, less enthusi-

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Crystalline estrogenic substances were provided by the Abbott Laboratories and by the Schering Corporation.

1. (*a*) Mortimer, H.; Wright, R. P., and Collip, J. B.: The Effect of the Administration of Oestrogenic Hormones on the Nasal Mucosa of the Monkey (Macaca Mulatta), Canad. M. A. J. **35**:503, 1936; (*b*) Atrophic Rhinitis: The Constitutional Factor and the Treatment with Oestrogenic Hormones, ibid. **37**: 445, 1937. (*c*) Mortimer, H.; Wright, R. P.; Thomson, D. L., and Collip, J. B.: Intranasal Administration of Oestrogenic Hormones in Constitutional Deafness, ibid. **40**:17, 1939.

2. Blaisdell, I. H.: The Use of Oestrogenic Substances in Atrophic Rhinitis, Laryngoscope **48**:699, 1938.

astically, by Soskin and Bernheimer.[3] The latter investigators expressed the belief that any beneficial result from estrogenic applications was due to a mucosal hyperemia consequent on local liberation of acetylcholine, since they obtained superior results with a solution of prostigmine methylsulfate.

Hamilton[4] has shown that androgenic material acts on the nasal mucosa in the same manner as estrogenic material. Progesterone may possibly have an opposite effect, since Wilson-Pepper and Royle[5] reported that it decreased nasal discharge in 1 case of rhinorrhea.

We have found in the present studies that estrogenic material stimulates and then depresses the activity of cilia lining the buccopharyngeal mucosa of the frog. Since the response was obtained within a few minutes after the application of the estrogenic substances in solution, it must have been due to an immediate effect of these substances. Furthermore, the reaction did not involve or depend on changes in the blood supply, because in our experiments the mucosa was stripped off and examined away from the rest of the body of the frog. Stimulation of ciliary movement was produced by a narrow range of concentration of the estrogens, and, since larger doses depressed ciliary activity, it appears that the dosage is an important factor in this form of topical therapy.

EXPERIMENTAL PROCEDURE

Our procedure was somewhat similar to that of Lucas and Henderson.[6] We stripped the mucosa off the posterosuperior surface of the buccal cavity and off the lining of the esophagus of pithed leopard frogs (Rana pipiens). The mucosa was pinned over a flat cork without being stretched. The cork was weighted and placed in a small evaporating dish, which was then filled with a 0.75 per cent solution of sodium chloride in distilled water until the surface of the saline solution was about 1 cm. above the strip of mucosa. To measure ciliary motion, we placed a small mustard seed on the mucosa and measured with a stopwatch the time required for the cilia to carry the seed a distance of 1 cm. A distance of 1 cm. was marked off on the edge of a slit cut in a piece of mica, which lay over the mucosa and was raised above it about 0.25 cm. by supporting pins at each corner.

Variation in Method.—We first ascertained the accuracy with which we could measure ciliary movement under these conditions and found that the results were quantitatively reproducible within a reasonable limit of error. For example,

3. Soskin, S., and Bernheimer, L. B.: Mechanism of Oestrogenic Effect on Nasal Mucosa in Atrophic Rhinitis: Successful Treatment with Prostigmine, Proc. Soc. Exper. Biol. & Med. **42**:223, 1939.

4. Hamilton, J. B.: Changes in Nasal Mucosa of Monkeys (Macaca Rhesi) and Humans by Male Hormone Substances, Proc. Soc. Exper. Biol. & Med. **37**: 366, 1937-1938.

5. Wilson-Pepper, J., K., and Royle, H.: Progesterone and the Nasal Mucosa, Brit. M. J. **1**:974, 1939.

6. Lucas, G. H. W., and Henderson, V. E.: The Initial Stimulation Phase in Narcosis: Experiments on Cilia and Embryos, Arch. internat. de pharmacodyn. et de thérap. **46**:465, 1933.

a series of eighteen consecutive readings on one strip of mucosa gave the following intervals in seconds for passage over 1 cm.: 28, 29, 28, 34, 23, 28, 29, 30, 26, 24, 31, 27, 32, 33, 28, 32, 27, 29. We repeated this procedure on 10 frogs and, to get an index of the variation, calculated in each case the mean, the standard deviation and the coefficient of variation proposed by the late Karl Pearson. The results are shown in table 1. The average coefficient of variation was 6.3, which is acceptable for quantitative biologic analysis of this type.

Determination of Factors Influencing Time of Survival of Ciliary Action.—We next investigated how long cilia remained active after the mucosa was removed from the frog and placed in a solution of sodium chloride. After a number of preliminary experiments it was found that there were several factors which affected the

TABLE 1.—*Variations Inherent in the Measurement of Measurement*

Specimen	Number of Trials	Mean, Sec. per Cm.	Standard Deviation, Sec. per Cm.	Coefficient of Variation
1	18	22.6	2.8	12.8
2	18	25.8	2.7	10.4
3	18	19.4	0.86	4.4
4	18	15.1	0.31	2.0
5	15	24.4	0.76	3.1
6	15	27.5	1.4	5.0
7	21	20.0	1.3	6.5
8	15	22.0	1.9	8.6
9	18	19.4	0.93	4.8
10	18	28.8	1.5	5.2

TABLE 2.—*The Interval Between Measurements and the Time of Survival of Cilia*

Interval, Hours	Time of Survival, Hours
24	<24
12	<12
6	6 to 12
3	9
1.5	4.5
0.75	4.5
0.50	4.5

period of movement, namely, the frequency of subjecting the cilia to measurement, the temperature of the immersion bath and the renewal of the saline solution.

The effect of frequency of measurement was studied by taking readings at intervals of from one-half hour to twenty-four hours on groups of 10 specimens each in a 0.75 per cent solution of sodium chloride at room temperature. In each group the number of hours elapsing before half the specimens became inert was noted, and these results are recorded in table 2. Ciliary movement survived longer if the cilia were not subjected to too frequent measurement.

The colder the solution of sodium chloride in which the cilia were immersed, the longer ciliary movement lasted. Thus, in one experiment with 10 specimens in a solution at 4 C. half of them did not become inert until after ninety-six hours.

The most important factor, however, appeared to be the replacement of the solution of sodium chloride with fresh solution at frequent intervals. When fresh solution of sodium chloide was substituted every one to three hours, measurements made every hour indicated that the cilia were all just as active at the end of twelve hours as at the beginning of the experiment. This experiment was conducted with the solutions at room temperature.

From these various considerations, we adopted as a general rule that readings be taken not more frequently than every half hour and that the solution of sodium chloride be changed at least every three hours.

NORMAL SPEED OF CILIARY MOVEMENTS

Lucas and Henderson [6] gave a wide range for the normal speed of ciliary movements in the buccoesophageal mucosa of the frog, between eighteen and sixty-nine seconds; they used small pieces of cork propelled by cilia, and apparently the distance carried was not the same for all specimens, but rather "from 1 to 2 cm." Gordonoff [7] quoted Lommel as finding that a particle of Lycopodium is passed along the ciliated mucosa of the trachea of a dog at the rate of 1 cm. in twenty-five to

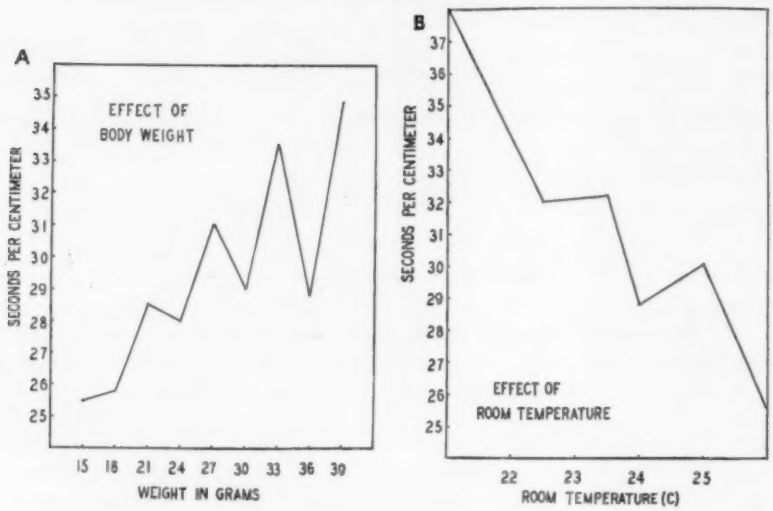

Fig. 1.—Charts showing: *A*, the effect of body weight on the rate of ciliary movement; *B*, the effect of room temperature on the rate of ciliary movement.

thirty seconds. The latter figure is approximately the same as the average figure which we have obtained from experiments on frogs.

From specimens of mucosa of 127 frogs, we found that the average rate of passage was 1 cm. in twenty-nine and one-tenth seconds and that the standard deviation of this average was four and two-tenths seconds. The normal rate of ciliary movement is, however, a value which is affected by a number of factors.[8] We have found that smaller frogs have

7. Gordonoff, T.: Der Expectorationsvorgang und seine pharmakologische Beeinflussung, Wien. klin. Wchnschr. **51**:289, 1938.

8. Negus, V. E.: The Action of Cilia and the Effect of Drugs on Their Activity, J. Laryng. & Otol. **49**:571, 1934. Proetz, A. W.: Nasal Ciliated Epithelium, with Reference to Infection and Treatment, ibid. **49**:557, 1934.

more active ciliary movements than larger frogs, and this is illustrated in figure 1 *A*, in which each point represents the mean of 10 specimens at least. Relatively small variations in room temperature may have a considerable effect on ciliary movements, and slight increases from day to day may increase the rate appreciably, as illustrated in figure 1 *B*, in which are plotted mean rates derived from measurements taken on groups of at least 10 specimens each. These various results indicated that all experiments must be rigidly controlled so that groups are compared at the same time and under the same conditions.

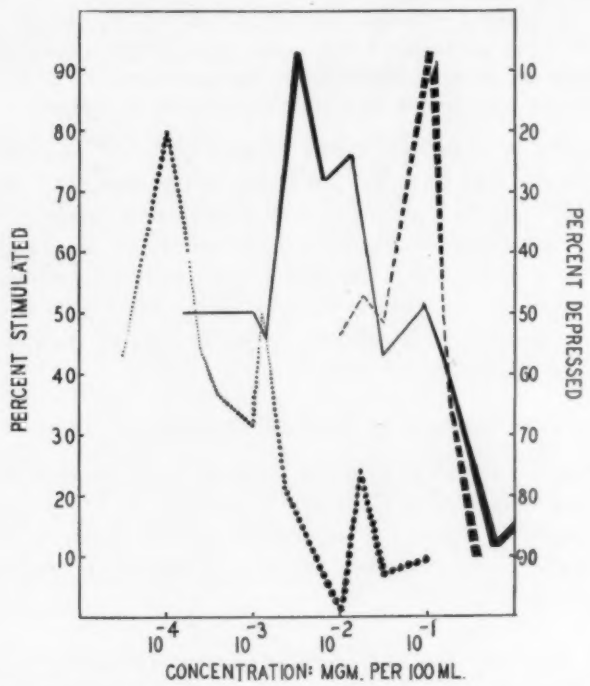

Fig. 2.—Chart showing the percentage of trials in which varying doses of estradiol (dotted line), estriol (solid line) and estrone (interrupted line) depress or stimulate ciliary movement.

EFFECT OF ESTRONE (THEELIN)

The final procedure, adopted to demonstrate the effect of estrone, was as follows:

Specimens of mucosa from 10 frogs were arranged in a 0.75 per cent solution of sodium chloride and left there for at least twenty minutes. Any mechanical adjustments, such as the changing of solutions, caused a disturbance in the ciliary movements, which did not reach an equilibrium for about twenty minutes thereafter. Two initial readings were taken on each specimen so equilibrated, and the saline solution was then replaced by a 0.75 per cent solution of sodium chloride

containing the concentration of estrone to be studied. Two readings were taken after the specimens had been in this solution one-half to one hour; the solution was then replaced with a 0.75 per cent solution of sodium chloride and the process repeated several times.

The mean number of seconds required for carrying the seed was calculated, and from these figures was found the percentage of trials in which estrone had increased or decreased the speed of ciliary movement. Since only occasionally were identical readings obtained in solution of sodium chloride and then in the estrone solution, it is obvious that 45 to 55 per cent of stimulations or depressions would be expected normally and that only variations beyond these are significant. To emphasize this, variations from 50 per cent of trials have been indicated by lines of increasing width in figure 2.

Estrone was added to a solution of sodium chloride in concentrations from 10^{-2} to 5×10^{-1} mg. per hundred cubic centimeters. To aid solution of the larger doses, the substance was first dissolved in a minimal volume of alcohol, the same volume of alcohol being added to the control solution of sodium chloride.

Concentrations of estrone below about 7×10^{-2} mg. per hundred cubic centimeters had no effect on ciliary movement, and doses above about 2×10^{-1} mg. per hundred cubic centimeters produced a marked depression. Concentrations between the relatively narrow range of about 7×10^{-2} and 2×10^{-1} mg. per hundred cubic centimeters produced a marked increase in the speed of ciliary movement. These results are illustrated in figure 2, in which each point represents the percentage of stimulations or depressions in at least twenty trials.

EFFECT OF ESTRIOL (THEELOL)

Proceeding in a similar manner, we found that crystalline estriol in concentrations between about 2×10^{-3} and 2×10^{-2} mg. per hundred cubic centimeters of solution of sodium chloride stimulated ciliary movement. Concentrations less than this had no effect, and higher concentrations were depressant.

EFFECT OF ESTRADIOL (DIHYDROTHEELIN)

The cilia were found to be most sensitive to estradiol. Ciliary movement was increased by concentrations of the crystalline substance between about 7×10^{-5} and 2×10^{-4} mg. per hundred cubic centimeters of solution of sodium chloride. Larger concentrations were depressant.

COMMENT

These experiments have revealed that cilia of the buccoesophageal mucosa of the frog are most sensitive to estradiol and least sensitive to estrone and that the sensitivity to estriol lies midway between. In minute amounts, too small to affect the hydrogen ion concentration (confirmed by measurement) or the electrolyte concentration, these estrogens first stimulate and then, in larger doses, depress ciliary movement. If we give the value of 1 to the stimulating power of estrone toward ciliary

movement, then estriol may be calculated to be twenty times as active as estrone and estradiol one hundred times as active.

This order of activity toward cilia does does not correspond to the order of estrogenic activity of these crystalline substances. If estrone is given an estrogenic activity of 1, estriol may be estimated as one one hundredth as active and estradiol as ten times as active as estrone. These figures for the relative estrogenic activity of the three estrogens have been calculated by approximations from the review by Kurzrok.[9]

A comparison of the relative ciliary stimulating powers of the estrogens with their various physical constants, reviewed by Allen,[10] also failed to reveal any other characteristic corresponding to the relative power to stimulate cilia.

Since cilia form an integral part of the protoplasm of the mucosal cells, the order of activity of estrogens on ciliary movement may reflect a corresponding order of activity on other functions of mucosal protoplasm. Among these other activities, we were especially interested in the power of estrogens to relieve ozena and atrophic rhinitis, which has been reported by the Montreal group.[1] If the present data have any application to the latter studies, they indicate that dosage is an important factor. Possibly the failure of Soskin and Bernheimer [3] to obtain results as good as those of the Montreal group may have been due to a difference in dosage, but, since Soskin and Bernheimer have given no figures on dosage, this cannot be confirmed.

The present data, based on their ciliary stimulating power, suggest that estradiol could be used in the lowest doses but that estriol would provide the widest and safest range of stimulating doses. From figure 2 it may be found that the maximal stimulating dose of estradiol is about five times the lowest stimulating dose, the maximal stimulating dose of estriol is ten times the lowest and the maximal stimulating dose of estrone is five times the lowest.

SUMMARY

After an investigation of factors which affect movements of cilia taken from the buccoesophageal mucosa of the frog, a method of assaying the effect of estrogenic substances on ciliary motion was worked out. Crystalline estrone, estriol and estradiol, dissolved in a solution of sodium chloride, stimulated ciliary movements when given in minute doses and depressed them when it was given in large doses. The range of doses producing stimulation when applied topically was about 7×10^{-2} to 2×10^{-1} mg. of estrone, 2×10^{-3} to 2×10^{-2} mg. of estriol and 7×10^{-5} to 2×10^{-4} mg. of estradiol per hundred cubic centimeters of saline solution.

9. Kurzrok, R.: The Endocrines in Obstetrics and Gynecology, Baltimore, Williams & Wilkins Company, 1937.

10. Allen, E.: Sex and Internal Secretions, ed. 2, Baltimore, Williams & Wilkins Company, 1939.

MODERN TREATMENT OF OTOSCLEROSIS

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Every form of therapy should preferably, of course, be based on pathogenesis; but when one lacks knowledge of the cause of a disease one has to be content with symptomatic treatment. So far as otosclerosis is concerned, symptomatic treatment has hitherto been the rule, even though recently in cases in which one hoped, *ex juvantibus,* to be able to trace the cause, some tentative efforts have been made at the alternative. To these trial measures, which so far have gained no greater importance, I shall have reason to return later. A rational effort to remove the symptoms consists in the establishment of an artificial labyrinthine sound fistula.

Anatomic investigations, in conjunction with those of a clinical nature, into the prevalent and severe aural disease called otosclerosis [1] have established that the central factor in this syndrome of progressively reduced hearing, often of a hereditary nature, with tinnitus, giddiness and paracusis willisiana, is an ankylosis of the stapes. Anatomic studies have also shown, however, that ankylosis of the stapes occurs not only in otosclerosis, with its peculiar transformation of the bony capsule of the labyrinth, but also in adhesive processes localized in the niche of the fenestra ovalis. Strangely enough, adhesive processes also occur with the formation of new bone, though it is of a different kind from that formed in otosclerosis. In most instances, processes of this kind are the result of recurrent catarrh or inflammation of the middle ear and generally lead, according to Wittmaack, to inhibition of the pneumatization of the temporal bone. Nowadays otologists generally claim that a roentgenogram of the cellular system of the ear will support the diagnosis of otosclerosis. There is as yet, however, no single symptom or sign in the clinical picture of otosclerosis, indeed, scarcely a combination of several symptoms and signs, by which the condition can be fully differentiated from an adhesive process. Yet, if all the criteria of otosclerosis are present, the diagnosis must be accepted. I shall explain my view more in detail. Hereditary disposition to adhesive processes is a common characteristic in certain families, commoner

After a paper read before the Oto-Laryngological Section of Svenska Läkaresällskapet, Oct. 27, 1939 (Svenska läk.-tidning. **37**:137-145 [Jan. 19] 1940).

1. A term selected by Politzer.

perhaps than that to otosclerosis. In its earlier stage a chronic adhesive process always seems progressive and particularly so when induced by chronic tubal catarrh. It may also take a protracted course. Spontaneous variations such as arise in the symptomatic picture of otosclerosis may occur in that of adhesive processes. An acute inflammation of the tympanum giving rise to adhesions around the stapes does not necessarily leave permanent signs in the cellular system. There is also the possibility that two such common diseases as I am dealing with here may affect the same ear. But surely, so it is argued, the adhesive process is, as a rule, not localized to the niche of the stapes alone; it immobilizes the whole of the ossicular chain. Instances of this are often seen, but one may also find the malleus stationary while the stapes is mobile. This is evident from the fact that in a case in which the malleus is apparently stationary a tympanic membrane prosthesis may greatly improve the hearing. Such an observation in the absence of a movable stapes would be inexplicable, since the sound before the application of the prosthesis would have had still better access to the round window. In recent years otologists have, contrary to their former practice, included under otosclerosis also those cases in which the mobility of the malleus has been more or less reduced. While Nager entirely disregarded the question of the mobility of the malleus in settling the diagnosis, Holmgren has even offered a hypothetic explanation of the ankylosis of the malleus as a result of that of the stapes. He also maintained, in a discussion on otosclerosis in the Oto-Laryngological Section of the Swedish Medical Society in 1939, that the differential diagnosis in many cases leaves the otologist in the lurch. The question then arises: As a safeguard against impaired chances for a future useful operation for otosclerosis, should one rule out all cases of adhesions in which there is reduced mobility of the malleus? Otologists are then at once confronted with the difficulty that up to the present there have been no means of exactly gaging the variations in movement exhibited by the malleus in persons of normal hearing. (I have taken up this technically troublesome question for investigation.) On the other hand, I am quite convinced that an artificial permanent sound fistula for ankylosis of the stapes is as effective in the case of adhesive processes as in that of otosclerosis. And in arranging a new acoustic route I can see no theoretic objection, in neutralizing also a possibly closed fenestra rotunda, to applying a second fistula, with its appropriate attributes.

In the latter half of the last century extensive attempts at evolving a rational form of treatment were made in various civilized countries, all with slight results or none. Such attempts were Kessel's extraction of the stapes, in 1876, and the following efforts at making up for the damage to the stapes window. After a few bold and unsuccessful

attempts at making a new window on the promontory, Bárány initiated, at about the same time as Jenkins, his fruitless series of sound fistulas on the horizontal semicircular canal. Later he shut off the tympanum from the retrotympanic region by free transplantation of a fatty flap, which stimulated the formation of a membrane. His idea was to protect the labyrinth from spread of infection from the tympanum. Concurrently he gained an air-filled space around the semicircular canal, after the fatty flap had been removed. I shall never forget the dramatic effect of opening the semicircular canal with a chisel—first, the terrible giddiness and then the striking rise of hearing. During the next few days the patient experienced great happiness in spite of the troublesome vestibular symptoms. Then hearing became gradually worse, and the patient's disappointment and despair were suggestive of the final stage of otosclerosis. One even heard of aggravations of the original deafness. I cannot say whether these were merely signs of the natural progress of the disease or were, in addition, influenced by the operation itself, as in Jenkins' cases. The most lasting improvements in hearing I can recall from this period, 1918 to 1919, extended over four months. Holmgren, who after Bárány took up the surgical treatment of otosclerosis, introduced improved optical and other instruments into the work, besides inspiring Sourdille, a French surgeon, to similar efforts. While Holmgren was content with making one or several permanent sound fistulas, Sourdille, in addition, carried out one or two surgical measures known as tympanolabyrinthopexy. The acoustically important detail in his operation was the removal of the head of the malleus for establishing greater mobility in the ossicular chain. From the younger Bárány's academic dissertation one learns that such an alteration also brings about augmented bone conduction. According to a statement in Sourdille's great work on otosclerosis, in 1935, he had observed improved hearing also in the ears which had not been operated on. He was speaking of a *réflexe oto-otique* and was wondering whether it was a question of some trophic or vascular effect. In the same work he has given the phenomenon the name *sympathie auriculaire* and has stated that the effect is of short duration, after which deafness may resume its progressive course while the ear on which operation was performed is keeping its level. In some cases, however, it seems as if the operation retards the progress of the disease in the other ear. When Holmgren, in 1939, confirmed Sourdille's statement that the ear on which no operation was performed also showed improved hearing, he reported that he had found the hearing in the ear which was operated on improved before he had opened the semicircular canal. Holmgren offered no explanation of these phenomena, but I suggested that they were probably due to the

skeletal vibrations set up by the chiseling. This assumption has since been sustained by the following experiment:

A mechanic aged 48 had a bilateral adhesive process or a so-called combined otosclerosis. The pneumatic cells were relatively well developed on both sides. He heard conversation on the less affected side at 2 to 3 meters; on the more seriously affected side he heard conversation at 2 to 3 decimeters and whispering—a couple of words *ad concham.* I explained the situation and asked him if I might carry out skeletal vibration, which possibly would bring about a temporary improvement of the hearing and, if worst came to worst, would perhaps afterward cause slight deterioration, although numerous explorations of diseased ears had produced evidence against this possibility. The patient smilingly underwent a skeletal vibration in the form of a feigned chiseling carried out on the more seriously affected ear by means of a center punch directed against the mastoid process in a 5 mm. incision through skin and periosteum. I imitated the strokes used in the process of chiseling, in groups of five. Hearing successively improved so that the patient was able to hear conversation at 3 meters, after which the treatment was terminated. For two days the patient was in a state of great excitement over his new hearing; on the third day, however, this rapidly deteriorated, and after four days his hearing was found to have returned to about its original level. One month later hearing was about the same; at any rate one was unable, either subjectively or objectively, to find any change from the original state.

Since then I have repeated this experiment in a classic case of bilateral otosclerosis, that of a business man aged 55, after extensive tests, including audiometric tests,[2] for sound frequencies and for speech. The result was entirely negative, and the patient's hearing immediately after the experiment, as well as months later, was exactly the same as before the experiment. Obviously, the results of such experiments in cases in which one is unable to gage the mechanical effect on the ear even by an approximate estimation of the mechanical resistance of the auditory changes, must be left to chance; i. e., they will differ from case to case. On the other hand, one must carefully consider whether cutting into the extremely hard bone labyrinth may not damage a sensitive internal ear, causing it to become the seat of post-traumatic degeneration. This might explain why the deafness after an operation for otosclerosis sometimes becomes rapidly worse.

Another question brought to the fore in 1939 by Prof. Max Meyer refers to the intralabyrinthine pressure in otosclerosis. Meyer maintained that Holmgren has proved that lowering of the intralabyrinthine pressure leads to improvement of hearing. On this assumption, according to his third report on the subject, in June 1939,[3] he is building up

2. The Aga-audiometer was the instrument used. Aulin and Nasiell: An Audiometer with Phonograph Records for Practical and Scientific Use, Nord. med. (Hygiea) **4**:3900, 1939.

3. Meyer, M.: La ponction sous-occipitale et son influence sur les hypoacousies dans l'otosclérose et l'atrophie labyrinthique, Ann. d'oto-laryng., 1939, p. 575.

new therapeutic experiments by drawing off, through a suboccipital puncture, a large quantity of cerebrospinal fluid. He also asserted that Sourdille held the same view, although this author in 1935 definitely refuted the idea. The latter author, as well as Wittmaack, also maintained that there is no certainty of an increase of perilymph or endolymph, i. e., of increased pressure in otosclerosis. He further stated that the perilymph is fairly copious when the disease is in an early stage and the hearing relatively good and that it is readily reproduced under such conditions; it is more scanty, however, when the disease is more advanced, and particularly is it said to be tardily reproduced in that stage. Now I shall examine Holmgren's proof more closely. Holmgren [4] stated that when exposing, in 1920, a fistula ante fenestram on the promontory, he noticed a fairly marked improvement in the hearing when a hair-fine jet of clear labyrinthine fluid came out of the fistula. About this he wrote in 1937:

Durch diese Beobachtung ist festgestellt, dass eine Dekompression der Labyrinthflüssigkeit Besserung des Gehörs bewirken kann. . . . Die Dekompression der Perilymphe, die hierbei zustande kam, kann meiner Meinung nach eine Rolle für die bei Anlegung der Schallfistel konstant und sofort eintretende Besserung des Gehörs spielen.

Dasselbe gilt bei Dekompression der Endolymphe. Wenn man bevor die Bogengangsfistel angelegt ist einem Teil der vorderen lateralen Wand des Saccus endolymphaticus freilegt, tritt in vielen Fällen—nicht in allen—sofort eine augenfällige, wenn auch nicht hochgradige Besserung des Gehörs ein.

(This observation proves that decompression of the labyrinthine fluid can bring about improvement of hearing. . . . Decompression of the perilymph, which thus took place, can in my opinion play a role in the constant and immediately occurring improvement of hearing after institution of the auditory fistula.

The same thing occurs with decompression of the endolymph. If, before the fistula is made into the semicircular canal, one uncovers the anterolateral wall of the saccus endolymphaticus, in many cases—not in all—an immediate, perceptible, if not very great, improvement of hearing takes place.)

It seems to me that Holmgren's conclusions are insufficiently substantiated. In exposing the preformed fistula by lifting away the mucoperiosteal graft on the promontorium he removed a mechanical obstacle to the transmission of sound pressure to the labyrinthine fluid whether this pressure passes from the round window to the fistula or vice versa. Tullio, professor of physiology in Bologna, in his monograph on the ear stated that Tyndall and other investigators have shown fluid jets to be sensitive to tones. Chichester Bell has further established that a fluid jet is influenced by all tones lower than its fundamental one. A jet of such fineness as that in question here might have a fundamental

4. Holmgren, G.: Zur Chirurgie der Otosklerose, Ztschr. f. Hals-, Nasen- u. Ohrenh. **40**:440, 1937.

tone of a pitch above the whole register of speech. Closer analysis of these details belongs to the expert physicist, but it can probably be deduced that the fluid jet might be so influenced by the sound pressure as to cause alterations of this to be transferred to the labyrinthine fluid in spite of the smallness of the opening.

By removing the relatively thick dura mater over the saccus endolymphaticus the operator is again eliminating an acoustic obstacle. It stands to reason that when the sound pressure thus directly strikes the wall of the saccus, the patient will hear better, provided, however, that the ductus endolymphaticus is not obliterated. According to anatomic examinations, this happens now and then, which explains Holmgren's statement that improvement in the hearing does not always take place. If an acceptable physical explanation has thus been given of the two phenomena described, nothing has thereby been said of the effect of reducing the pressure. Of this nothing is known. So far as I am aware, neither Holmgren nor any one else has offered any acceptable reason why in otosclerosis the labyrinthine pressure would be increased. Even the normal pressure of the fluid in the labyrinth is not known. Against the theory of a rise of pressure is the fact that the vulnerable nervous mechanism in the cochlea may remain intact even in advanced otosclerosis.

Although Meyer, as I have shown, has based his new therapeutic method for otosclerosis on faulty premises, his statement regarding the excellent results obtained in hearing is sufficiently sensational to cause one to test his method. Before saying anything about the results obtained I shall make a few comments on Meyer's work. After having declared in his third report that Holmgren's observation, as related previously, is evidence in proof that the improved hearing resulting from operations for otosclerosis is partly due to reduction of the intralabyrinthine pressure, he went on to state that he had changed his mind regarding labyrinthine pressure and now believed that the perilymphatic pressure is normal, since the cerebrospinal pressure is normal, but that the endolymphatic pressure is lowered. Discussing the different components, obstacles to conduction and so-called neurogenous components, in advanced otosclerosis, Meyer seems to have overlooked Markmann's proof that the combined reduction of hearing associated with this disease can be explained solely by ankylosis of the stapes, since this author in a case of that nature found the perceptive organ quite normal. This old observation of Markmann also agrees with the discovery of recent years that an obstacle to conduction may reduce perception of high-pitched tones (Bekesy, Crowe, Guild and Shambaugh Jr.). This fact I have also been able to verify in a recent case of salpingitis, in which, in addition, Rinne's test was positive. Both deafness and tinnitus disappeared

immediately on catheterization. In Meyer's [5] last report, there are graphs of hearing in which it is shown that the great improvement began only five and three months after the drawing off of the fluid. If the improvement arose as a result of reduced pressure in the labyrinth—which, of course, soon enough must become equalized—one cannot help wondering why it did not come immediately after the withdrawal of fluid. The effect on the hearing is also said to be obtained in cases in which the condition is neurogenous.

After having made myself familiar with the details of suboccipital puncture in Professor Antoni's neurologic department where he and his assistants afforded me every facility, I have tried Meyer's treatment in 7 cases. Meyer draws off 35 to 50 cc. of cerebrospinal fluid. I did not always succeed in getting that amount. In 1 case I stopped at 16 cc., as the patient, a woman aged 47, then began to suffer pains in one ear. The pain stopped after a while, which was regarded as a promising sign. After all, there was some effect on the diseased organ. The case was a typical one of bilateral otosclerosis with all its criteria in regard to history, hearing and roentgenograms. Hearing for conversation was —27 decibels in both ears (pure air conduction, with room acoustics eliminated). The patient did not hear any better, but an unpleasant complication developed the following day, in the form of headache and stiff neck, with subfebrile temperature. Kernig's sign was not present. Subsequently giddiness supervened, and the patient remained in a nursing home for a month, after which she went for a holiday. She returned just over two months after the operation and then felt quite well. Objectively her hearing was roughly the same; subjectively it was improved; tinnitus had ceased. Evidently this was a fairly poor result, as the positive effect can be ascribed to rest and medication. In 3 other cases of bilateral otosclerosis cerebrospinal fluid was withdrawn in amounts of 35, 44 and 22 cc. The hearing of all the patients remained unimproved; 1 of them was last treated in April, the others in May. From a woman aged 46, who had had progressive bilateral reduction of hearing for the past two years, whose response in the Rinne test was positive and who had neurasthenia, 13 cc. of cerebrospinal fluid was withdrawn. The tapping, which in all cases had been done with the patients in the sitting position, had in this case to be interrupted for psychic reasons. In this single case there was great improvement in the hearing, which, however, I ascribe to the rest and sedatives prescribed. The case remainds me of that of a Finnish singer whom I had under treatment many years ago in collaboration with Professor Brühl, of Berlin, Ger-

5. Meyer, M.: Eine neue Anschauung über die Schwerhörigkeit und vielleicht ein neuer Weg zur ihrer Behandlung, Acta oto-laryng. **27**:1, 1939.

many, and Professor Burger, of Amsterdam, Holland. The diagnosis was: surditas bilateralis neurotica. After two months' stay in the country, the patient's hearing, formerly much impaired, was completely restored. In a case of posttraumatic combined severe bilateral deafness, in which, on my suggestion, Professor Antoni tried cisternal puncture but on account of difficulty in getting the needle to pass the integuments of the neck performed lumbar puncture instead with the patient in the recumbent position, although 50 cc. of fluid was withdrawn, no improvement in the hearing was obtained. After the patient had had a prolonged rest in the country the troublesome tinnitus disappeared, but hearing remained as before. Tinnitus has subsequently returned. It is evident that my experience with these cases does not encourage me to further trials of the treatment.

In the same number of the *Annales d'otolaryngologie* in which Meyer published his third report, referred to previously, there is a review by another author under the title "La surdité hormonale."[6] A number of workers are referred to here who have tried to find the connection between otosclerosis and endocrine disturbances. The assumption of such a connection is based on the known fact that otosclerosis has shown some connection with pregnancy. And since the bony changes in the capsule of the labyrinth bear some relation to calcium metabolism, which is regulated by the parathyroid glands, several endocrine glands have been considered as being involved, in a dozen cases. The report of the fourth of these cases is typical of the reasoning of this author. A woman aged 32 had since the age of 26 suffered from progressive impairment of hearing. After an abortion in 1937 her hearing became considerably worse. Her mother became deaf at the age of 55 and a brother at 20. The Wassermann reaction was positive in 1937. Titration of urine for hormones showed: estrogen, 30 international units; gonadotropin, 40. The patient had deafness of the labyrinthine type. The treatment was intensively antisyphilitic with, in addition, administration of solution of parathyroid, 30 cc., and an estrogen, 10,000 international units per cubic centimeter, five times a month. Great improvement in hearing resulted. In 1938, the hearing became worse. Treatment was resumed.

One cannot help asking why the deafness of a syphilitic patient should be called hormonal; what proof is there, or even indication, that it would not have receded after merely antisyphilitic treatment?

Apart from this literature, there is reason to hope that endocrine research will throw fresh light on the causes of otosclerosis. Until more research has been done in that field, and for advanced stages a great deal more research, otologists will have reason to stick to the efforts of building a new line of sound conduction to the labyrinth which still functions.

6. Predescu-Rion: La surdité hormonale, Ann. d'oto-laryng., 1939, p. 554.

According to a report in 1939 by Holmgren and Nager in the Oto-Laryngological Section of Svenska läkaresällskapet, the former has lately made sound fistulas on the semicircular canals of monkeys, and the ears were, after some time, sent to Nager for microscopic examination. It was then found that all the fistulas had closed. Similar experiments were made by Sourdille on patients on whom he had operated both once and twice. This would seem to prove that the establishment of a permanent fistula on a semicircular canal remains as doubtful a procedure today as in 1919, when I had the opportunity of following Professor Bárány's endeavors. That Sourdille, as well as Holmgren, can record lasting improvement in the hearing does not alter the fact that the permanency of the fistula, despite all well intentioned efforts, is still a mere chance. Nevertheless, these operators on the labyrinth have gained considerable ground; they have shown in hundreds of cases that operating on a noninflamed labyrinth is not such a dangerous undertaking as was formerly believed.

It stands to reason that under the circumstances many an otologist has become more and more pessimistic regarding the possibility of establishing the wished-for labyrinthine fistula. I should like to remind those colleagues that it took nearly three hundred years to develop an acceptable method and technic for the drum prosthesis. In view of this, it would not be surprising if it took thirty years or more to attain permanent artificial sound conduction by way of a labyrinthine fistula. At any rate, for my own part, I feel convinced that the solution of the problem is close at hand. My theories regarding this will be published as soon as animal experiments have provided a solution to the problem.

A colleague, who recently died and who had suffered from otosclerosis, said to me once: "You may know a great deal, you otologists, but you are no good at curing deafness." His tone was bitter. Depressed, I kept silent, since I had nothing to offer him. Like many another deaf person, he did not want to make use of a hearing apparatus. It is true enough that the present therapy of deafness leaves much to be desired, but the outlook seems good. Otologists have the greatest possibilities of influencing obstacles to conduction. Statistics already show these to be more numerous than the inaccessible injuries to the perceptive apparatus. The number of cases in which deafness due to injury to the perceptive apparatus is diagnosed steadily diminishes with the increase in ability to apply new physical and physiologic experiences to the art of diagnosis. If to the present therapeutic arsenal are added the proper hearing prostheses in their full application and, within the near future, means of artificial, permanent labyrinthine conduction for patients with otosclerosis and adhesive processes, there will no longer be so great a percentage of hard of hearing people who need leave the otologist's

consulting room with the verdict, nowadays so common: "Nothing to be done." Not only for his clientèle, but for the otologist himself, these are happy prospects, the realization of which I hope I shall live to see.

SUMMARY

I have mentioned the fact that the causes of otosclerosis are unknown, and referred to recent attempts to find *ex juvantibus* an endocrine origin of the disease. The starting point of these efforts is the knowledge of the pathologic anatomy of the disease and its relation to pregnancy.

Attempts at rational treatment of the disease by establishing a sound fistula in the labyrinth as a compensation for the fixation of the stapes have hitherto met with failure. The cause of this failure is that proper mechanicoacoustic sound conduction was never established. Holmgren and Sourdille misinterpreted their observations in connection with their operative researches. A shifting has taken place of late in the diagnostic criteria of otosclerosis, with the result that new difficulties have arisen as to the differential diagnosis between otosclerosis and adhesive processes in the middle ear. In my opinion, however, these difficulties are of no practical importance, for I consider permanent artificial sound conduction to the labyrinth just as effective for fixed stapes in the adhesive process as for the same component in otosclerosis.

The results of my tests of the method by which Max Meyer was recently reported to have obtained splendid improvement of hearing in patients with otosclerosis, i. e., the suboccipital withdrawal of a considerable quantity of cerebrospinal fluid, have been negative. These results are well in accord with my observation that Meyer's work is founded on the faulty conclusions arrived at by Holmgren, i. e., that decompression of the labyrinthine fluid in patients with otosclerosis has been proved to increase hearing.

ACUTE LARYNGOTRACHEOBRONCHITIS

ANALYSIS OF SIXTY-TWO CASES WITH REPORT OF AUTOPSIES IN EIGHT CASES

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INTRODUCTION

Acute laryngotracheobronchitis is an infection of the respiratory tract, occurring most frequently in children under 2 years of age and characterized usually by primary inflammation of the pharynx, with later involvement of the larynx and trachea and extension to the bronchi. A sticky, gummy exudate produces variable respiratory obstruction, with severe symptoms of toxicity. The cause has not been determined definitely, but many organisms obtained from the laryngeal and tracheal cultures have been considered as causative factors. Recently, it has been suggested that a virus is the primary invader, preparing the way for secondary bacterial invasion, especially by the hemolytic streptococcus.

Greenwood [1] stated: "Acute laryngitis in infants is an inflammatory disease of the laryngeal intima, usually secondary to an acute upper respiratory infection, and generally associated with pathological changes in the lower respiratory tract." Jackson has divided acute inflammations of the larynx, per se, into (*a*) specific acute laryngitis and (*b*) nonspecific acute laryngitis. Acute specific laryngitis is an infective disease, with a definite course and a self-limited termination. Acute nonspecific

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Read before the American Academy of Ophthalmology and Otolaryngology, Cleveland, Oct. 10, 1940.

1. Greenwood, G. J.: Acute Laryngitis in Infants, Illinois M. J. **75**:52 (Jan.) 1939.

laryngitis is due to causes other than infection and does not follow a regular course.

We agree with Evans,[2] who spoke of acute laryngotracheobronchitis as of paramount importance to otorhinolaryngologists, pediatricians and general practitioners. The fact that the most expert efforts often fail to change the fatal trend of this malady has served in the past to make some physicians despair of attempting treatment. It is, therefore, extremely important that any measure which seems promising should be given a thorough trial, even though its mode of action is not entirely clear. The problems pertaining to treatment have been discussed by many physicians. The mortality still is high,[3] but without adequate facilities and competent nursing it would be higher.[4]

Acute laryngotracheobronchitis is by no means a new clinical entity. However, the marked increase in its incidence in the Eastern section of the country during the fall and winter of 1939-1940 has brought it again to our attention.

The purpose of this paper is to define terms, giving the corresponding clinical picture; to show the different stages and factors as the process develops, involving the various anatomic areas; to discuss the differential diagnosis and treatment; to present reports of 8 autopsies and, finally, to give a survey of 62 cases of acute laryngotracheobronchitis encountered at the Essex County Hospital for Contagious Diseases from 1936 to 1940. An effort has been made to evaluate the various methods of treatment, both medical and surgical. Especially has it been our effort to try to judge or evaluate the results obtained by intubation and tracheotomy and by the use of moist cool air. This will be taken up in more detail in the discussion of treatment.

We shall give a comparison of our results with those reported statistically by other authors (table).

Our series of cases comprises both mild and severe types of laryngotracheobronchitis. Mild laryngotracheobronchitis when given prompt and effective treatment may never reach the severe stage, in which surgical intervention is necessary.

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2. Evans, M. G.: Acute Laryngotracheobronchitis, Ann. Otol., Rhin. & Laryng. **48**:216 (March) 1939.

3. Smith, W. J.: Acute Laryngotracheitis in Children: Study of Forty-Three Cases Occurring at Children's Hospital, Los Angeles, in Epidemic Form, During Winter of 1933-1934, Arch. Otolaryng. **23**:420 (April) 1936.

4. Clerf, L. H., and Putney, F. J.: Peroral Endoscopy, Arch. Otolaryng. **31**: 979 (June) 1940.

We agree with Brennemann [5]

> that a sharp differentiation according to the precise nature of the involvement in a given case is not always a simple matter, especially as one must find its exact place in a series of manifestations that range in severity all the way from simple catarrhal laryngitis to rapidly fatal fulminating laryngotracheobronchitis.

We are not classifying separately those cases in which tracheotomy, intubation and bronchoscopic aspiration were required or those in which the patients died, but, as will be seen, we have included cases in which the clinical diagnosis was laryngotracheobronchitis but in which surgical procedure was not necessary.

HISTORICAL DATA

MacCready [6] gave a detailed report on the history of acute laryngotracheobronchitis, with a complete bibliography. In 1928 Baum,[7] reporting a series of 24 cases, brought out that in his experience intubation gave better results than tracheotomy. In 1936 Gittens,[8] reporting a series of 38 cases, emphasized the fact that the mechanical obstruction was more serious than the type of infection. In 1933 Richards [9] made the first extensive pathologic study of the condition. This was followed in 1938 [10] by another most interesting article, in which he discussed the possible use of sulfanilamide.

In 1939 Evans [11] reported a few cases in which he used a bacteriophage to liquefy the bronchial secretions. Apparently this was successful. In 1939 Galloway [12] reported 10 cases and advocated postural drainage, to assist the patient in expelling the secretion from the bronchial tree. In 1940 Felts,[13] reporting a series of 19 cases,

5. Brennemann, J.; Clifton, W. M.; Frank, A., and Holinger, P. H.: Acute Laryngotracheobronchitis, Am. J. Dis. Child. **55**:667 (April) 1938.

6. MacCready, P. B.: Acute Laryngotracheobronchitis, Connecticut M. J. **4**:123 (March) 1940.

7. Baum, H. L.: Acute Laryngotracheobronchitis, J. A. M. A. **91**:1097 (Oct. 13) 1928.

8. Gittens, T. R.: Laryngitis and Tracheobronchitis: Reference to Nondiphtheritic Infections, Tr. Am. Bronch. Soc., 1936, p. 122.

9. Richards, L.: Fulminating Laryngo-Tracheo-Bronchitis, Ann. Otol., Rhin. & Laryng. **42**:1014 (Dec.) 1933.

10. Richards, L.: A Further Study of the Pathology of Acute Laryngo-Tracheo-Bronchitis in Children, Tr. Am. Laryng., Rhin. & Otol. Soc. **44**:372, 1938.

11. Evans, M. G.: Acute Laryngo-Tracheo-Bronchitis, Ann. Otol., Rhin. & Laryng. **48**:216 (March) 1939.

12. Galloway, T. C.: Postural Treatment of Acute Laryngotracheobronchitis, J. A. M. A. **112**:1566 (April 22) 1939.

13. Felts, G. R.: Acute Infective Laryngotracheobronchitis, J. Oklahoma M. A. **33**:9 (March) 1939.

recommended the use of a room in which the atmosphere is kept supersaturated with moisture.

During the fall and winter of 1939-1940 we used a room in which the atmosphere was supersaturated with moisture and maintained at a temperature of 70 F. at all times, as well as additional moisture in the oxygen tent, with gratifying results. Davison,[14] however, must be given the credit for first bringing this type of treatment to the attention of the members of the medical profession in a paper, "Some Observations on the Control of Temperature and Humidity in Oxygen Tents," read before the Eastern Section of the American Laryngological, Rhinological and Otological Society at Pittsburgh in January 1940.

In all, then, over 300 cases in which the condition was so severe that the patients required hospitalization have been reported since 1918.

ANALYSIS OF CASES

The 62 cases of acute laryngotracheobronchitis herein reported were observed at the Essex County Hospital for Contagious Diseases. Over one-half the patients had a history of previous cold or infection of the upper respiratory tract, and the predominating organism was Streptococcus. Forty of the patients were males and 22 females; 47 were under 3 years of age, while the ages of the others extended over the first decade. There were 45 cures and 17 deaths. Two of the patients who died were moribund on admission.

We are of the opinion that intubation alone is certainly not the procedure of choice.

Intubation alone was performed on 8 patients, and only 1 recovered.

Tracheotomy alone was performed on 4 patients, with 2 recoveries, i. e., with a mortality of 50 per cent.

It is interesting, however, that with a combination of intubation and tracheotomy, which was done in 16 cases, 11 recovered (68 per cent recoveries, or 32 per cent mortality).

We feel that intubation tides a patient over until treacheotomy can be done. Intubation is a deciding factor in the reaction of dyspneic patients from respiratory shock, except in cases in which there is supraglottic swelling, when tracheotomy must be done at once.

Of our 29 patients whose dyspnea had to be relieved by some mechanical means, 26 were children under 3 years of age. During 1939, 16 children were tracheotomized. Five of these died, giving a

14. Davison, F. W.: Treatment of Acute Laryngotracheobronchitis, Arch. Otolaryng. **32**:321 (Aug.) 1940.

mortality rate of 31.25 per cent for this group. It is our belief that tracheotomy is the operation of choice and should be done as soon as possible.

Sulfanilamide was given in 20 cases, with 13 recoveries. Sulfapyridine (2-[paraaminobenzenesulfonamide]-pyridine) was given in 3 cases, with 2 recoveries. In 15 cases the two drugs were given in combination with some operative help, with 7 recoveries. In 8 cases sulfanilamide was without mechanical aid. All the patients recovered.

At the Essex County Hospital for Contagious Diseases it is routine procedure to give sulfanilamide to all patients suspected of having acute laryngotracheobronchitis, until the report of the laryngeal culture has been made, after which appropriate treatment is instituted. It is possible that in many cases in which sulfanilamide and sulfapyridine were given the disease was in a mild form.

Mortality Among Patients with Acute Laryngotracheobronchitis

Author	Cases	Gross Mortality, per Cent	Tracheotomies	Deaths	Mortality from Tracheotomy, per Cent	Dates
Gittens [8]	32	39	20	12	80	1936
Smith [3]	43	9.3	12	4	33	1936
Baum [7]	24	41	17	9	53	1928
Richards [9]	28	40	23	12	50	1937
Felts [13]	19	21	17	4	23	1940
Orton	62	28	20	7	35	1940
Galloway [12]	10	20	10	2	20	1939
Davison [14]	17	11.7	9	1	11.1	1940
Brennemann [5]	45	42.2	24	10	41.7	1938

The aforementioned figures may be compared with those in 38 cases in which neither drug was used. In the latter group we had 29 recoveries. In the cases in which recovery occurred, Streptococcus was the predominating organism.

We believe that, although sulfanilamide and sulfapyridine are not specifics for this disease, they seem to have some value.

Of the 15 patients whom we placed in a room supersaturated with moisture and maintained at a temperature of 70 F., 10 recovered. We are of the opinion that this form of treatment is a deciding factor in lowering the mortality rate of this disease.

CAUSATION

Acute laryngotracheobronchitis is an inflammation of the mucous membrane of the larynx, trachea and bronchi, usually preceded by symptoms of some infection of the upper respiratory tract, ordinarily rhinitis. In the absence of fever and a preceding cold the determination of the etiologic factor is sometimes difficult, as brought out by Johnson.[15]

15. Johnson, M. C.: Acute Laryngotracheobronchitis in Infants, Arch. Otolaryng. **17**:230 (Feb.) 1933.

The condition may, however, be primary in the larynx or in the subglottic region. Occasionally it follows the aspiration of a foreign body of vegetable origin. The disease is more prevalent among infants and young children, although adults are not immune. Barry [16] emphasized the fact that this condition occurs also in adults. In his case there were pure cultures of Staphylococcus aureus haemolyticus, with an abundant crusting and suppurative infection of the entire respiratory tract. Direct examination of smears of material taken from the larynx and trachea usually reveals a mixed infection of staphylococci, streptococci and pneumococci. In some cases pure cultures will be found. As a rule, the disease is far more severe in children under 2 years of age, probably on account of the smallness of the subglottic area. The younger the child, apparently, the more severe is the infection. Of our 62 patients, 47 were under 3 years of age. No specific organism has yet been proved to be the etiologic agent of this disease. Most writers have found Streptococcus to be the predominating organism. Of our 45 cases in which cultures were made of material from the larynx, we found Streptococcus in 11; Streptococcus and Staphylococcus in 13; Streptococcus and Pneumococcus in 10; Staphylococcus in 4; Pneumococcus in 2; Streptococcus, Staphylococcus and Pneumococcus in 4, and Staphylococcus and Pneumococcus in 1.

It has been suggested that a filtrable virus may play a part in this disease, preparing the ground for secondary invaders. However, there has been no proof of this. The low blood count observed in some of our cases suggests a virus disease.

PATHOLOGY

Clerf [17] stated: "The gravest pathologic finding is the marked subglottic swelling; . . . . Acute laryngotracheobronchitis remains one of the most serious conditions that has to be dealt with in children."

The pathologic findings depend on the causative organism. In all types of infection, especially during the early stages, there is an inflammatory reaction. In our series 51 patients showed gross pathologic changes in the larynx, epiglottis, vocal cords, arytenoid, subglottic area and trachea.

16. Barry, A.: Membranous Laryngitis and Tracheitis with Staphylococcus Aureus Hemolyticus Infection, Ann. Otol., Rhin. & Laryng. **37**:960 (Sept.) 1928.

17. Clerf, L. H.: Acute Laryngotracheobronchitis, Arch. Otolaryng. **29**:722 (April) 1939.

In cases in which Streptococcus is the infecting organism, the inflammatory reaction is associated with marked fiery redness and swelling of the subglottic and tracheal regions, together with a pronounced velvety appearance of the tracheal mucosa. The true vocal cords, however, may show comparatively little reaction. As the disease progresses, necrotic gangrenous pseudomembranes may be seen extending down to the

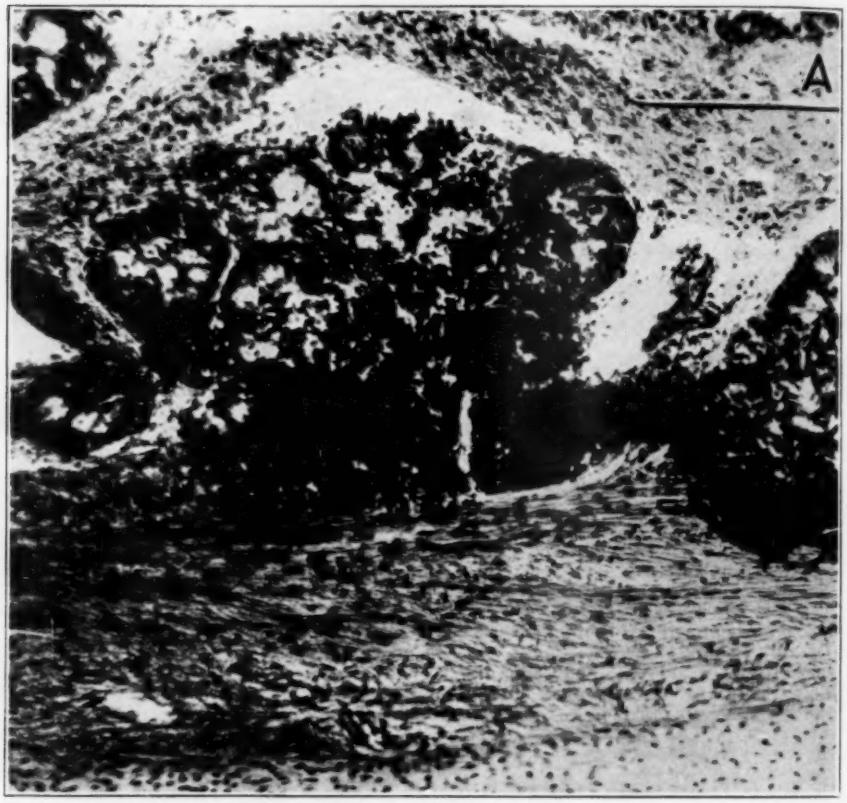

Fig. 1.—Edema of the submucosal structures (*A*) of the trachea.

tracheal bifurcation and into the main bronchi. Early in the course of the disease the subglottic swelling causes mild dyspnea. Later, the obstructive gangrenous pseudomembrane is responsible for more pronounced dyspnea, often extreme and leading to a surgical emergency.

If, however, the causative agent is Staphylococcus, the inflammatory reaction of the tracheal and pharyngeal mucosa is practically always accompanied with the formation of dirty gray, thick, tenacious mucous

plugs, which tend to dry and crust and rapidly cause marked progressive obstructive symptoms. In our series 6 patients had a pseudomembrane in the larynx. Frequently, early in the course of the infection, some of these crusts are seen covering the vocal cords and obstructing the glottis. Removal of the tracheal and bronchial crusts does not reveal any necrosis of the tracheal mucosa. This, however, is frequent late in the course of the streptococcic type of the disease.

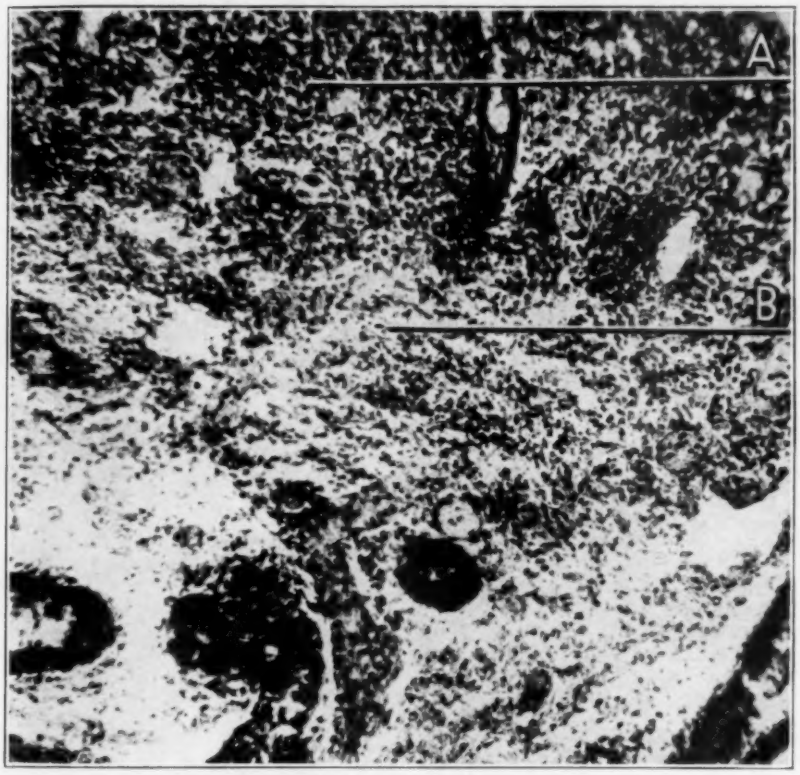

Fig. 2.—Purulent exudate (*A*) adherent to the mucosa. Edema and polymorphonuclear infiltration of the underlying tissues (*B*) of the trachea.

Bacteriologically, we found in our series the following distribution of causative organisms:

11 cases, Streptococcus alone
13 cases, Streptococcus and Staphylococcus
10 cases, Streptococcus and Pneumococcus
4 cases, Staphylococcus
2 cases, Pneumococcus
4 cases, Streptococcus, Staphylococcus and Pneumococcus
1 case, Staphylococcus and Pneumococcus

45

In the blood picture we observed nothing startling other than a marked shift to the left, due to the infection. This was noted in the vast majority of the patients. However, late in our study we attempted to ascertain the carbon dioxide content of the blood. This was done in only 6 cases—too small a number from which to make definite deduc-

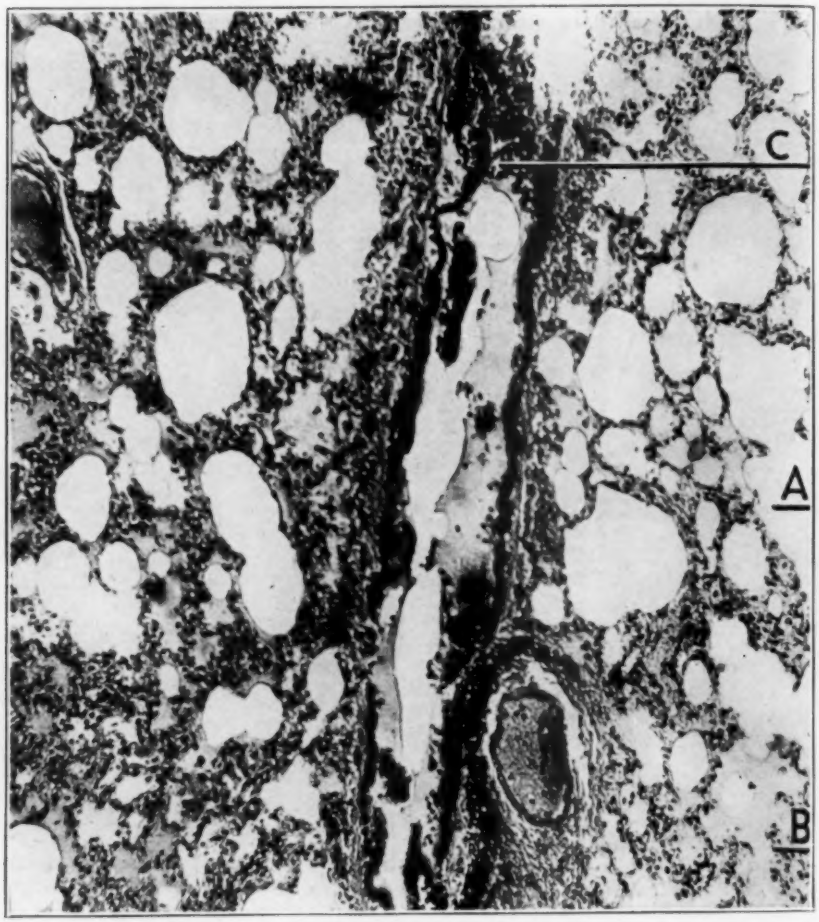

Fig. 3.—Emphysema (*A*). Atelectasis and interstitial pneumonitis (*B*). Purulent exudate in terminal bronchus (*C*).

tions. In 3 cases, in which the carbon dioxide content was 41 or below, the patients died. The other 3 patients, with a carbon dioxide content of the blood above 42, survived.

Autopsies on 8 patients who died from acute laryngotracheobronchitis will be given in detail. A summary of these 8 autopsies shows that the external nares had some crusts and mucopurulent material, evidence of an infection of the upper respiratory tract. The pharynx

and tonsils were inflamed. The mucous membrane lining the larynx, trachea and large bronchi was swollen, hyperemic and edematous. In nearly its entire extent, it showed a thick, somewhat adherent mucopurulent exudate, which separated easily from the underlying tract and left no gross area of erosion.

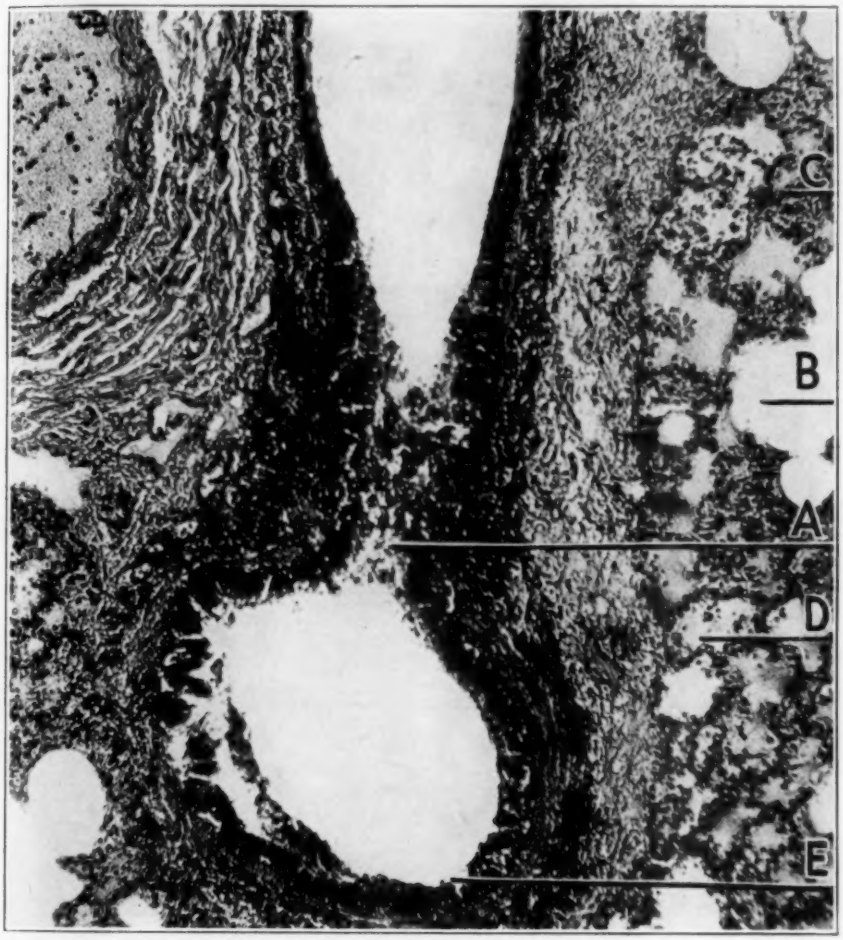

Fig. 4.—Mucus plug (*A*) in a small bronchus, which contains, in addition, many cellular elements. Emphysema of alveolar spaces (*B*). Atelectasis and interstitial pneumonitis (*C*). Mucoid exudate in small alveoli (*D*). Ulceration and partial destruction of lining epithelium of bronchiole (*E*).

Laryngoscopic examination of a patient with acute laryngotracheobronchitis reveals a deep red swollen subglottic mucosa, often covered with yellowish crusts or a mucopurulent material. At times the swell-

ing is so great that little airway remains, and this is further obstructed by the outpouring of the thick tenacious secretion. Occasionally there is also extensive supraglottic edema, especially of the epiglottis, a condition which may be detected easily by digital examination.

Bronchoscopic examination shows a thick tenacious secretion in the trachea, often obstructing the openings of the bronchi. The tracheal

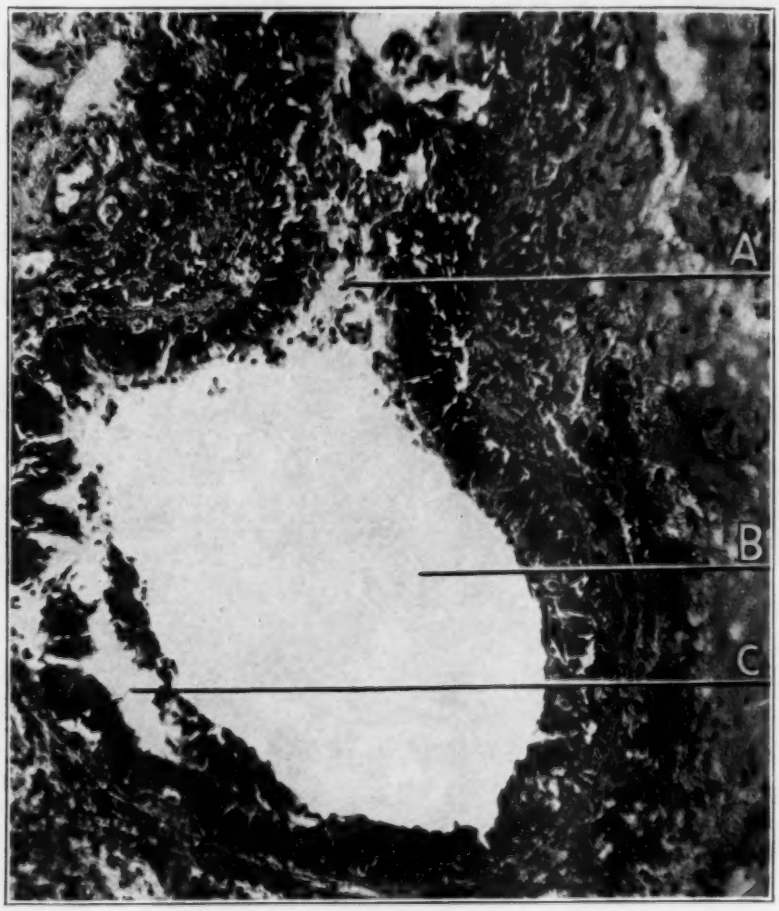

Fig. 5.—High power magnification of a section of a lung, showing exudate obstructing the bronchus (*A*); distention of the distal segment (*B*), and destruction of the lining epithelium (*C*).

mucosa is red and swollen, but with further progress of the disease, as Brennemann [5] stated:

> the intensely red mucosa has been lost and is replaced throughout the tracheal tree by an exudate with a dry, glazed, gray surface. It is thick enough to cause a decided narrowing of the tracheal lumen and in some instances to cause

such a narrowing that even the smallest aspirator cannot be passed where previously a small bronchoscope had entered.

The parenchyma of the lungs on cross section is seen to be somewhat congested and to contain small amounts of thin sanguineous fluid, but there is no gross evidence of consolidation, and the lungs seem to be well aerated. The smaller bronchioles are red and are congested with a moderate amount of glairy bloody material. The glands at the

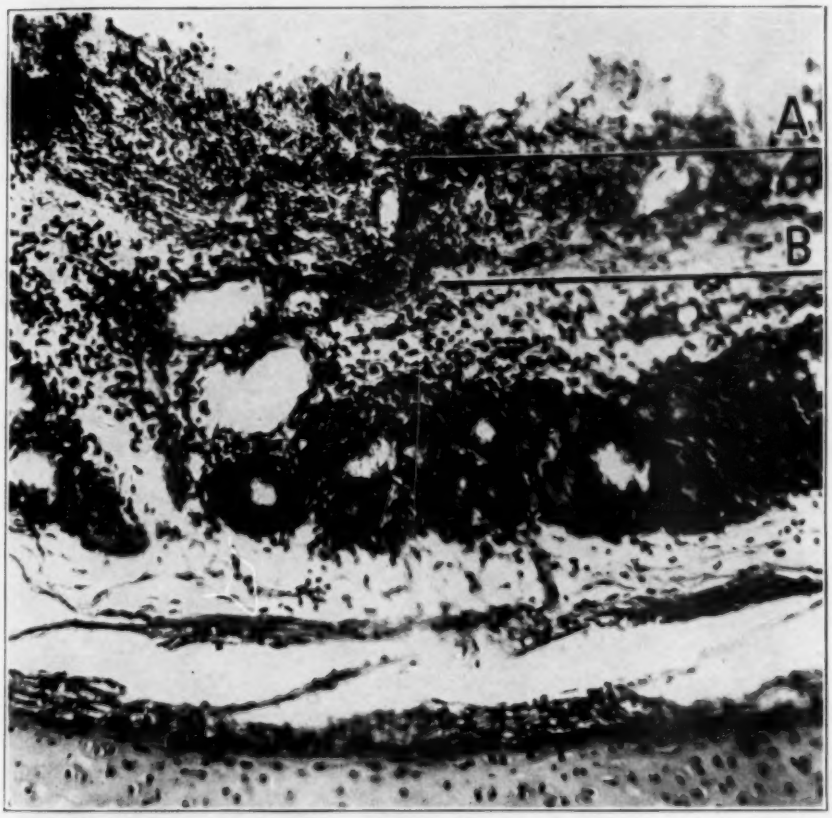

Fig. 6.—Bronchus, showing purulent exudate (*A*) adherent to the bronchial wall, with total destruction of the underlying epithelium (*B*).

hilus of the lung and along the large bronchi and trachea are enlarged and somewhat congested. Some specimens show, scattered throughout all lobes of both lungs, small areas of patchy consolidation. In these areas, the surface appears red and congested, and the smaller ramifications of the bronchial tree contain tenacious mucopurulent material and their lining membrane is red and edematous. On account of this the larynx, trachea and bronchi show a reduction of caliber. An elastic purulent exudate gives the appearance of a pseudomembranous lining.

Microscopic examination of sections from the lung shows that the small bronchi are filled with a purulent exudate and that the walls are diffusely infiltrated with polymorphonuclear leukocytes. In some areas the lining epithelium has partially degenerated, and in others it is

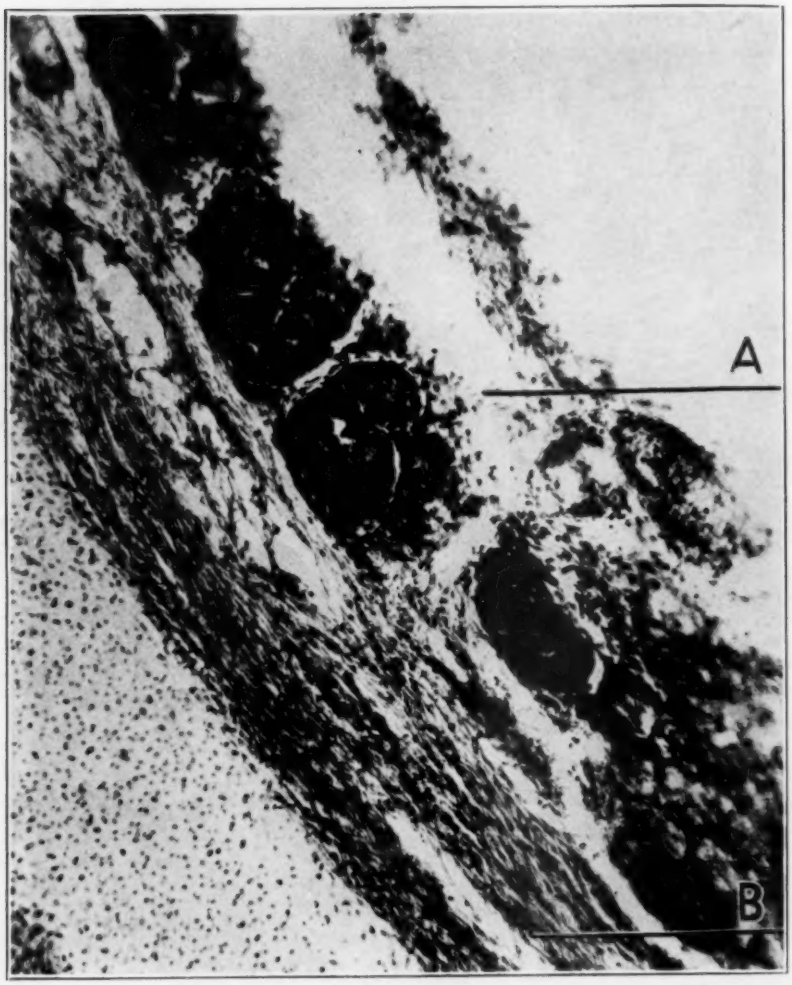

Fig. 7.—Bronchus, showing inflammatory lining wall with edema and destruction of the epithelial layers beneath (*A*); edema and polymorphonuclear infiltration of the subepithelial layers (*B*).

entirely missing. These structures are edematous, and the blood vessels are congested. Some of the alveoli surrounding these bronchi are distended with purulent exudate, consisting chiefly of myriads of polymorphonuclear leukocytes and fibrinous material, together with degen-

erated epithelial cells. In some places the alveolar walls are edematous and thickened, and in others there is partial collapse of the alveolar structure. In some specimens microscopic examination shows the lining membrane to be absent, as if there were many small ulcerations. The smaller bronchi are filled with masses of inflammatory exudate,

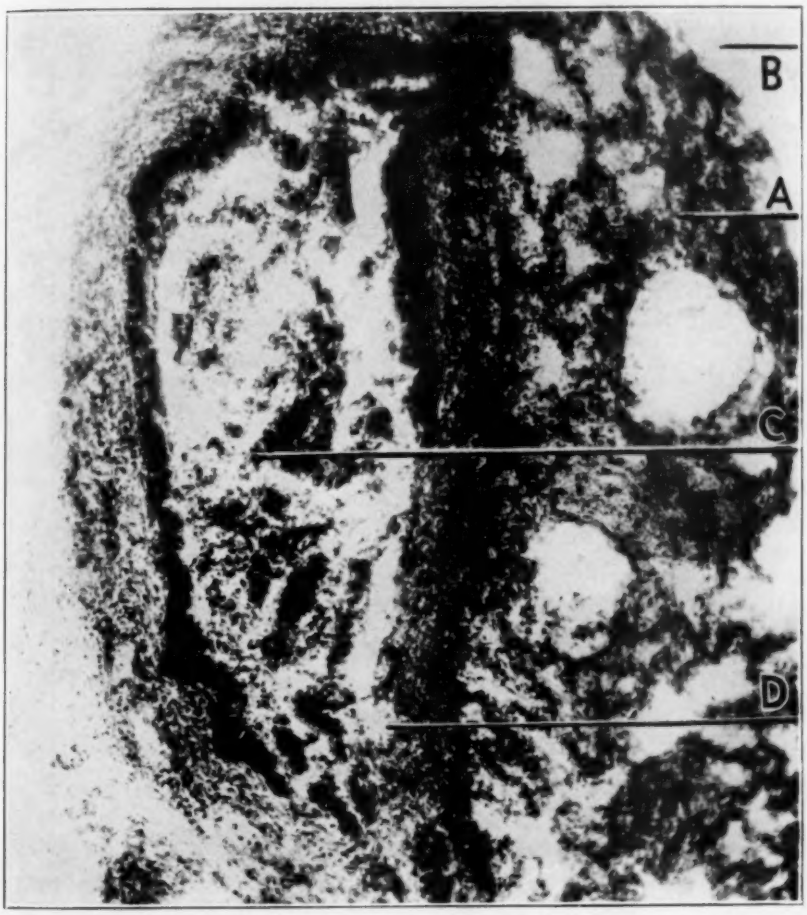

Fig. 8.—Lung with atelectasis and thickening of the alveolar wall, with polymorphonuclear infiltration (*A*); emphysema (*B*); purulent exudate in the bronchus (*C*), and destruction of the bronchial epithelium (*D*).

consisting chiefly of intact and partially degenerated leukocytes and fibrinous material. The walls of the immediately adjacent alveoli are somewhat thickened and show evidence of inflammation. In other specimens there are partial collapse and atelectasis of the alveolar structure, presumably from the exudate within. In still others the alveolar spaces

are filled for the most part with a collection of polymorphonuclear leukocytes, and in some regions there are small patches of hemorrhage. In other regions the alveoli are within areas containing gross hemorrhages, the blood vessels being distended with blood.

In addition to confirming the bronchoscopic observations, autopsy has shown atelectatic and emphysematous areas, as well as bronchopneu-

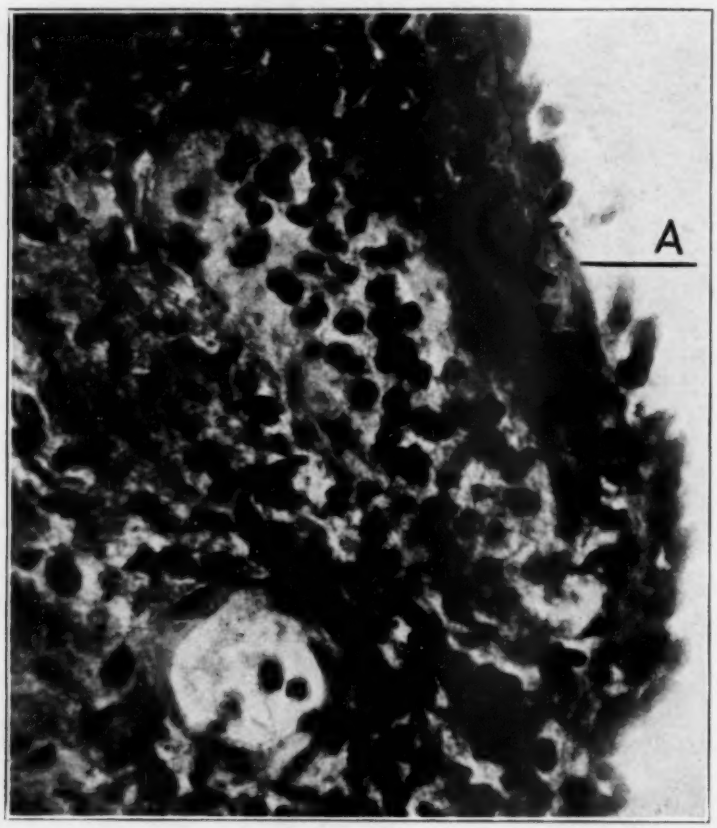

Fig. 9.—High power magnification of a section from a bronchus, showing ulceration and partial destruction of the lining epithelium (*A*).

monia. These findings attest further to the severity of the infection, with its accompanying mucopurulent secretions and the resultant obstructive symptoms.

Further studies of the pathologic changes in the tracheobronchial tree and the trial of systemic treatment with drugs are necessary to produce a sizable reduction in mortality.

SYMPTOMS

The disease may be ushered in by an infection of the upper respiratory tract, with its associated symptoms, soon followed by fever. This occurred in over half of our cases, with a history of illness for an average of three days before admission to the hospital.

The patients are acutely ill, with temperature ranging from 102 to 106 F., rapid pulse, marked respiratory distress and a toxic appearance. In well over 50 per cent of the cases studied, hoarseness appeared early and was preceded or followed by a croupy cough, denoting subglottic involvement. Later it was accompanied by dyspnea, with retraction, pallor and sometimes, but not often, cyanosis. Dyspnea with retraction was noted in 47 cases.

In streptococcic infections the elevation of temperature is usually higher than in the staphylococcic invasions. At this stage, auscultation of the chest may reveal failure of air to enter the lungs because of obstruction of the bronchi by dry crusted mucous plugs.

As the disease progresses, the dyspnea becomes more severe, with marked retraction of the suprasternal space, the supraclavicular space and the epigastrium and marked depression of the intercostal spaces occurs, accompanied with pallor, restlessness and sometimes, but not often, cyanosis.

The final picture, as so ably described by Richards,[9] is "a ghastly spectacle of death by asphyxiation and suffocation" or, as I have termed it, peripheral asphyxia. Such is the usual course of the disease unless some prompt courageous treatment is instituted. Sometimes even then all efforts are futile.

DIAGNOSIS

Acute laryngotracheobronchitis is by no means a new clinical entity. However, the marked increase in its incidence in the section served by the Essex County Hospital for Contagious Diseases during the fall and winter of 1939-1940 has brought it again to our attention.

Early diagnosis is just as important as in any other disease if one expects to lower the mortality. From the number of cases reported here, it is obvious that laryngotracheobronchitis is a serious disease, and the need for early diagnosis is evident when one considers the diagnoses made by the physicians who sent the patients in our series to the hospital:

Tonsillitis	1
Diphtheria	27
Croup	25
Foreign body	1
Whooping cough	5
Scarlet fever	1

Since the diagnosis of diphtheria tops the list, we think we should state, as we justly may, that laryngeal diphtheria can now be considered a rare condition. The onset may resemble that of laryngotracheobronchitis. However, nondiphtheritic laryngitis usually presents marked

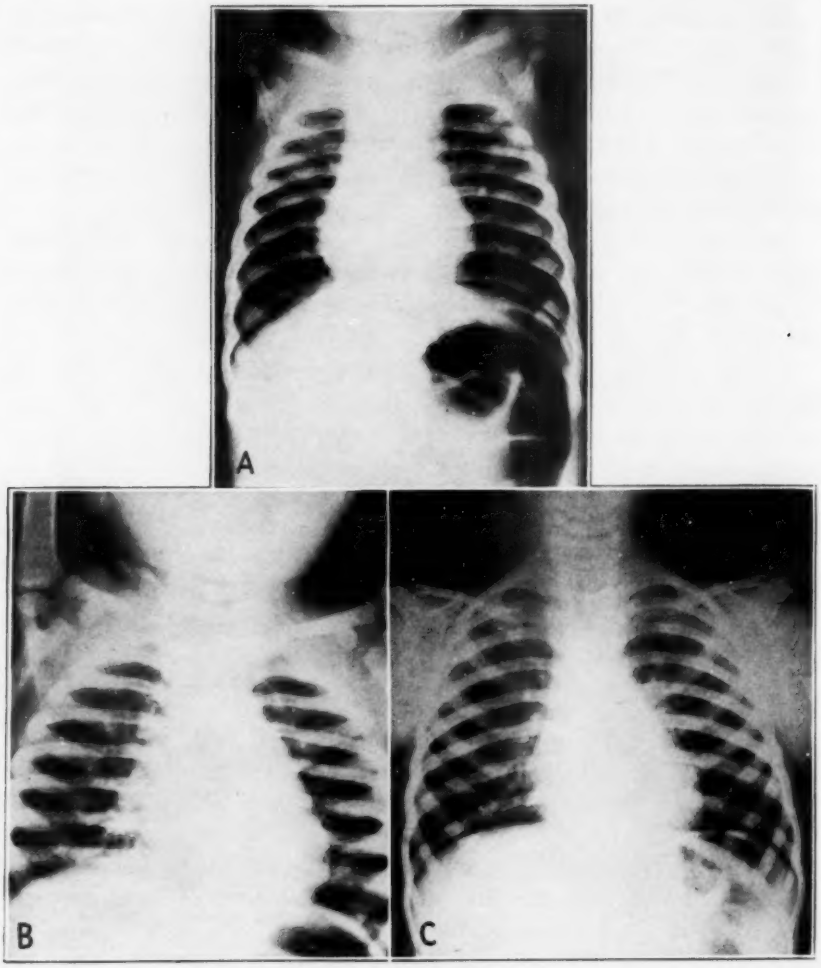

Fig. 10.—*A*, roentgenogram of T. B., aged 5 months. There is slight bilateral enlargement in the supracardiac region, with pressure on the trachea. *B*, roentgenogram of L. A., aged 15 months. Both hili are increased in density. The bronchial markings are slightly infiltrated throughout both lungs. There are no signs of pneumonia. *C*, roentgenogram of E. H., aged 2½ years. Both hili are increased in density. The bronchial marking are slightly infiltrated throughout both lungs. There are no signs of pneumonia.

subglottic changes, whereas laryngeal diphtheria, although it may be primary in the larynx, is more commonly an extension from the

pharynx. Direct smears from the laryngeal membrane,[18] which should be taken in all cases, invariably reveal diphtheria bacilli. In such cases, even though diphtheria antitoxin is administered on suspicion of diphtheria, it is imperative to supplement this treatment by either intubation or tracheotomy. This is also extremely important in the treatment of acute laryngotracheobronchitis and therefore should be done early.

The purpose here is to define terms, to give the corresponding clinical picture, to show the different stages and factors as the process develops, involving the various anatomic areas, and finally to discuss the differential diagnosis.

Terms, Synonyms and Definitions.—1. Croup—Any affection of the larynx in children, characterized by difficult and noisy respiration and hoarse cough.

(*a*) Catarrhal croup, of acute catarrhal laryngitis: simple inflammation of the mucous membrane of the larynx.

(*b*) False croup, laryngitis stridulosa, spasmodic laryngitis: simple inflammation of the mucous membrane of the larynx, accompanied by night attacks of closing of the glottis, causing inspiratory stridor.

(*c*) Membranous croup, or pseudomembranous croup: inflammation of the larynx in which there is a pseudomembranous exudate on the vocal cords.

(*d*) True croup, or membranous croup: inflammation of the larynx caused by the diphtheria bacillus.

(*e*) Spasmodic croup, or laryngismus stridulus (no laryngitis): spasmodic closure of the glottis, lasting a few seconds, followed by noisy inspiration. The terms imply that there is no inflammation, as in tetany, in breath-holding spells, in convulsions and in the paroxysms of whooping cough.

2. Acute laryngotracheobronchitis—inflammation of the mucosa of the larynx, trachea and bronchi.

The three terms "catarrhal laryngitis," "spasmodic laryngitis" and "laryngotracheobronchitis" by definition most accurately describe the pathologic condition responsible for the clinical picture. Because of common usage, however, the selection of the term "spasmodic laryngitis" instead of "spasmodic croup" is for the most part a technicality.

In the early stages of the process it is impossible to make a differential diagnosis of these clinical entities. In many instances even after the patient has improved it is indeed difficult to know just where to draw the line. An accurate and detailed history is helpful. The fact

18. Hart, V. K.: Streptococcic Laryngitis, Ann. Otol., Rhin. & Laryng. **36:** 761 (Sept.) 1927.

is that a condition beginning as catarrhal laryngitis or as spasmodic laryngitis may remain that or may progress to laryngotracheitis or laryngotracheobronchitis.

Illustrative Cases.—The first case which we shall describe will serve to illustrate the clinical picture of catarrhal laryngitis.

R. H., a boy 3½ years of age, was admitted to the hospital on April 22, 1940. The chief complaint was difficulty in breathing. The illness had started some six days previously, with an infection of the upper respiratory tract. On the morning of admission there were difficult breathing and brassy cough. The child's temperature was 101.8 F., his pulse rate 120 and his respirations 29. Physical examination revealed a well developed and well nourished boy, acutely ill and in respiratory distress. His color was good, though there was slight circumoral pallor and his cheeks were flushed. There was a slight mucopurulent discharge from the nose. The soft palate and the pharyngeal walls were inflamed. There was no exudate or pus. Laryngoscopic examination showed the entire laryngeal mucosa red and infected and the cords slightly thickened, but no exudate or membrane was present. There was no enlargement of the cervical glands. Examination of the chest revealed evidence of respiratory embarrassment, and there were suprasternal and infrasternal retractions with each inspiration. The intercostal spaces also were retracted slightly. Auscultation revealed harsh breath sounds. No rales were heard. When the child cried or was disturbed, there was a slight croupy brassy inspiratory sound. Further examination revealed no positive findings. On the following day, April 23, the patient's general condition was markedly improved and there was only slight respiratory embarrassment. The blood picture showed the hemoglobin content 61 per cent, erythrocytes 3,720,000 and leukocytes 12,800 per cubic millimeter, with polymorphonuclears 82 per cent and lymphocytes 18 per cent. There were 4 band forms and 78 segmented forms. The urine was acid; the specific gravity was 1.025; it showed albumin (a trace) and acetone (4 plus); the diacetic acid and dextrose content were normal, and microscopic examination gave negative results.

On April 24 the child was breathing easily, with no retraction, although his voice continued to have a slightly croupy sound and he had an occasional hoarse cough. On April 25 his temperature rose to 100.4 F. However, he had no subsequent elevation of temperature or any further symptoms. He was discharged from the hospital on April 30.

The clinical picture of spasmodic laryngitis is illustrated by the second case.

B. C., a boy 4 years of age, was admitted to the hospital on April 4, 1940 at 2 a. m. He had been suddenly awakened from his sleep because of difficulty in breathing and a brassy cough. Physical examination revealed a well developed boy of healthy appearance in acute distress due to difficulty in getting air into his lungs. There were suprasternal, infrasternal and intercostal retractions. The child did not appear toxic or fatigued. However, there was some cyanosis of his lips and finger nails. The temperature was 99 F., the pulse rate 155 and the respirations 43. The tonsils were enlarged. The fauces and pharyngeal walls were moderately inflamed. No membrane, exudate or pus was present. Auscultation of the chest revealed a loud harsh sound over the larynx, trachea and large bronchi. No rales were heard. The blood and urine were normal. The child was given 2 drachms (7.76 Gm.) of syrup of ipecac, after which he vomited

and was somewhat relieved. At 4 a. m. the temperature was 98.6 F., the pulse rate 115 and respirations 40. By 8 a. m. the respiratory rate had dropped to 25. From this time on the child made an uneventful recovery.

In cases such as that just described laryngoscopic examination gives negative results except during the spasm, at which time there is discoloration of the laryngeal mucosa, the color varying from lavender to bluish, as is expected in any spasm.

The characteristics of laryngotracheobronchitis are illustrated in the third case.

P. C., a 23 month old child, was sent to the hospital on Feb. 18, 1940 with the diagnosis of croup and obstructive pneumonia. The history stated that the child became ill suddenly during the night two days before, with difficulty in breathing.

Examination revealed a well nourished male infant, acutely ill and struggling to breathe. There were slight cyanosis of the lips and nails and pallor about the face. There were suprasternal and infrasternal retractions. The voice was fairly good. There was some dried secretion about the nares. The tonsils and pharynx were only moderately congested. The breath sounds were harsh, but no rales were heard.

Direct laryngoscopic examination revealed an acute inflammation of the laryngeal mucosa, with subglottic edema. No membrane or exudate was present. The temperature was 103 F., the pulse rate 144 and the respirations 40. The urine showed albumin (a trace) and acetone (2 plus). The blood picture was as follows: hemoglobin content 65 per cent; erythrocytes 3,540,000 and leukocytes 12,400 per cubic millimeter; polymorphonuclears 70 per cent; lymphocytes 28 per cent, and monocytes 2 per cent. After three hours' observation, as the child showed no response to treatment and became exhausted, a low tracheotomy was done, with immediate relief. Shortly after the tracheotomy the breathing was easy, the color was a healthy pink and the child fell asleep. By the fourth postoperative day the temperature had become normal, and the child made an uneventful recovery.

Laryngotracheobronchitis with a less fulminating course is noted in the fourth case.

S. W., an infant of 9 months, was admitted to the hospital at 1 a. m. on Oct. 19, 1939. The history was that four days before, at 4 a. m., he had been seized with an attack of croup. During the following two days he had alternate periods of difficult breathing and of apparent relief. During the next two days he had no relief. Physical examination showed a well developed child with difficult respiration, who appeared markedly fatigued. There were suprasternal and infrasternal retractions and a loud laryngeal crow. The skin was pale. The pharynx was moderately inflamed, but no exudate or membrane was present. Laryngoscopic examination revealed a red swollen laryngeal mucosa, with a large amount of greenish exudate. The exudate was aspirated, and a specimen for culture was taken from the larynx. The breath sounds were harsh, but no rales or dulness was discerned. The temperature was 102.4 F., the pulse rate 170 (questionable) and the respiratory rate 65. At 9 a. m. the same day the child was no better; thus, some operative procedure was necessary. Intubation with a one month O'Dwyer tube produced considerable relief. During the next five days the infant

was fairly comfortable, but some toxemia persisted, as shown by the variation of temperature from 101 to 103 F., with a downward trend. Likewise, the pulse rate fluctuated from 140 to 160 and the respirations from 40 to 50.

On admission the blood picture was as follows: hemoglobin content 62 per cent; erythrocytes 3,360,000 and leukocytes 13,700 per cubic millimeter; polymorphonuclears 66 per cent; lymphocytes 26 per cent; monocytes 6 per cent and plasma cells 2 per cent. This picture remained practically the same until two days after operation, when the leukocyte count was 23,650. Repeated urinalyses gave negative results. On Oct. 24, after having worn the intubation tube for six days, the child coughed up the tube and immediately became dyspneic and cyanotic. The tube was reinserted, and he experienced relief. A few hours later a low tracheotomy was performed. There was a moderate reaction, the temperature rising to 103 F. However, by the third postoperative day he appeared on the way to an uneventful recovery. On Dec. 1 the tracheotomy tube was removed, and on Dec. 6 the patient was discharged from the hospital with the wound healed.

In a résumé of the 4 cases cited here, we can say that the physical signs of acute laryngotracheobronchitis are as follows:

A well developed person has an infection of sudden onset, preceded by or accompanied with an infection of the upper respiratory tract. Usually the tonsils and pharynx are congested, with some glandular involvement. As a rule, the heart action is of good quality, though in some patients the pulse is irregular. As the infection increases, there is involvement of the larynx, which is indicated by hoarseness or a croupy cough with increased respiratory difficulty and retraction, at first slight, then rapidly becoming worse. The patient is critically ill and toxic, becoming restless, with pallor and later cyanosis. The picture is one of rapidly progressing respiratory asphyxia.

TREATMENT

The attending physician should bear in mind that every child with acute laryngeal obstruction is potentially a victim of acute laryngotracheobronchitis until proved otherwise. Too often this so frequently fatal disease is diagnosed as diphtheria, spasmodic laryngitis or catarrhal laryngitis, and only when the child is near death does the physician realize the gravity of the condition.

The efficient care of patients with this disease depends on a well trained hospital personnel, consisting of a readily accessible and competent bronchoscopist, experienced residents on call at all times and interns and nurses especially trained in the management of patients with this disease. It is essential that these patients be under constant observation night and day, for a change may occur suddenly, with a fatal outcome unless proper treatment is instituted immediately. It is our opinion that any child with continued signs of laryngeal obstruction should be sent to a hospital which has a personnel capable of handling this condition properly, rather than treated in the home.

On admission to the hospital, the patient is seen by the resident physician, and when definite laryngeal obstruction is noted, a laryngoscopic examination is done and material for culture taken from the larynx. This laryngoscopic examination is helpful in several ways. It enables one to determine whether or not a membrane suggestive of

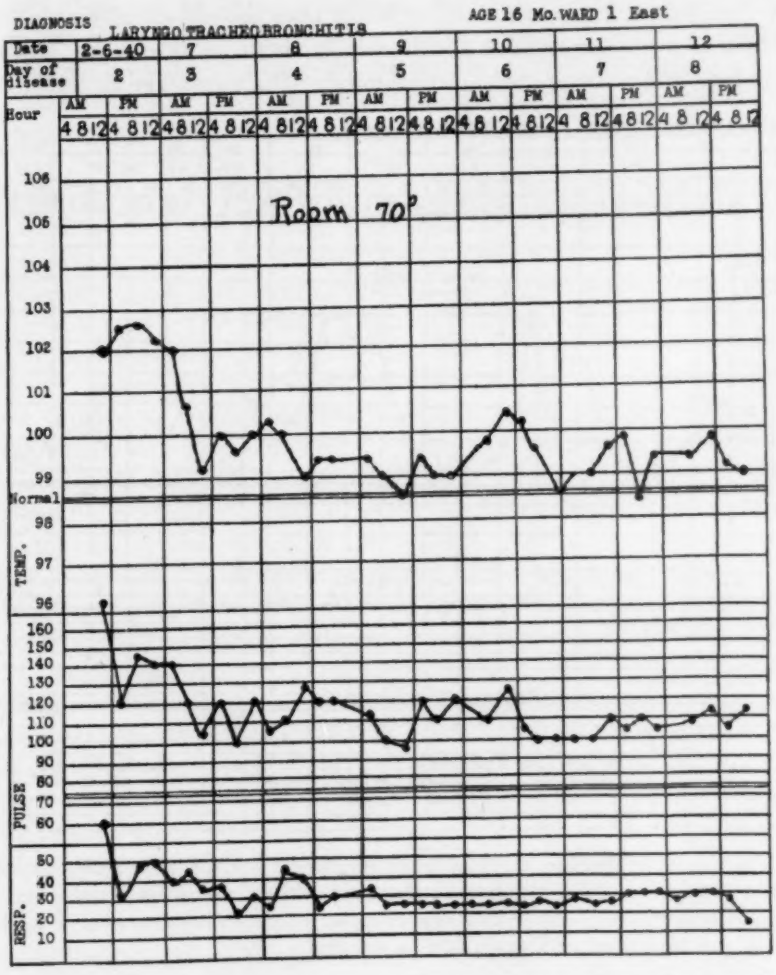

Fig. 11.—Chart showing the drop in the temperature of a patient aged 16 months after he was placed in a humidified room.

diphtheria is present. Material can be taken from the larynx for culture to determine the presence of diphtheria bacilli and organisms of other types, and also the amount of swelling present in the subglottic area can be observed.

If the patient's condition is such that operative relief of the obstructive symptoms is not immediately necessary, he is placed in a room the

air of which is supersaturated with moisture and maintained at a temperature of about 70 F. At the Essex County Hospital for Contagious Diseases we are able to obtain this moisture by letting hot water run into the tub in the bathroom adjoining the patient's room. More recently, we have used a Walton humidifier and found it satisfactory. We feel that an entire room is far better than the old croup tent, as the patient is more comfortable and is able to obtain more oxygen, and the watching and nursing care are facilitated. The advantage of the super-

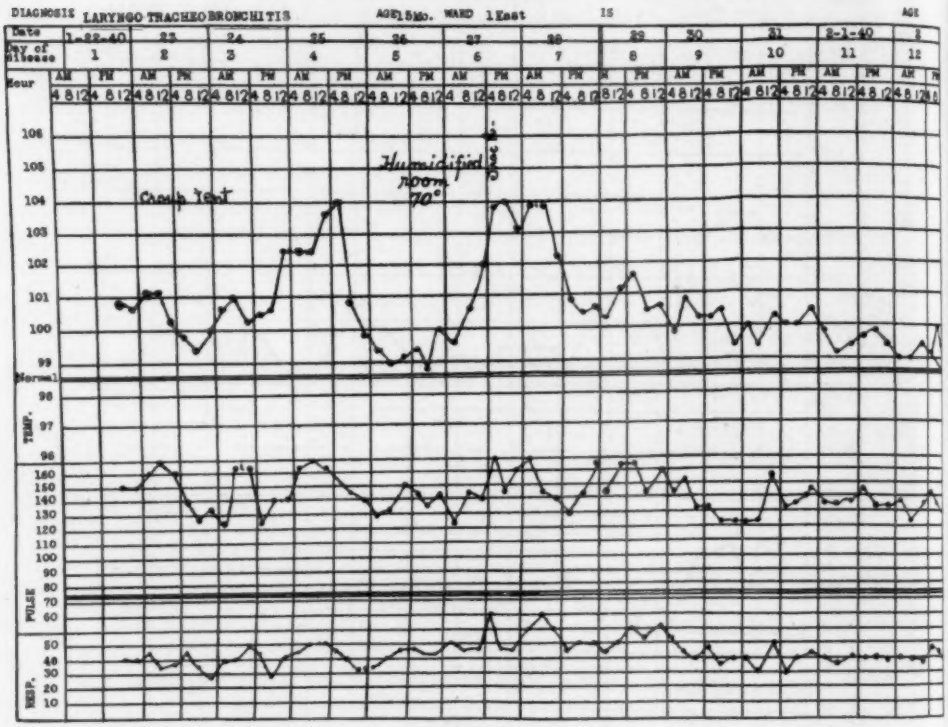

Fig. 12.—Temperature chart of a 15 month old patient with acute laryngotracheobronchitis. Note the drop of temperature after the patient was placed in the humidified room. After this improvement in the child's condition a tracheotomy was performed with practically no reaction.

saturated air is that it makes the patient more comfortable, enables him to breathe more easily and prevents loss of water. All this helps to keep the gummy tenacious exudate from drying and crusting and to liquefy it, so that it may be expelled more easily.

That inhalation of medicated vapor has any value in the treatment of this disease is doubtful, however time honored its use.

It has been our custom to give iodides in some form.

The use of opiates and belladonna is strongly condemned. Opiates further dull the cough reflex and produce an insidious restfulness, which sometimes is the harbinger of death. Belladonna and atropine serve to make the secretion one wishes to liquefy only drier.

An adequate supply of body fluids is necessary for the patient. Usually these can be given in sufficient amounts by mouth, but it may be necessary to administer them by clysis or intravenously. Usually soft and liquid foods can be given by mouth in adequate amounts.

When it becomes apparent by increasing dyspnea that the patient is not improving with these palliative measures, resort to some operative procedure must be considered. Greater respiratory distress manifests itself by increased restlessness, by suprasternal and infrasternal retractions and stethoscopic evidence that little air is entering the lungs. When the respiratory effort has been so great that the patient has become exhausted, restlessness decreases and ashen pallor, rather than cyanosis, occurs. The patient, apparently resigned to his fate, seems to give up the struggle for life.

Every patient with sufficient laryngeal or tracheal involvement to cause audible stridor on exertion is consdiered in the danger zone.[19] If the stridor is noticeable when he is resting, tracheotomy should be considered. If it is audible when he is asleep, the operation is urgently required. In a vast majority of our cases this condition was present on admission of the patient to the hospital. When dyspnea becomes urgent, immediate attention in the form of intubation or tracheotomy is required. The question may arise as to what operative procedure to employ. Aspiration through the bronchoscope may give temporary relief, but usually this will have to be repeated often, and it requires the constant services of a bronchoscopist. Intubation may be done quickly and usually gives immediate relief unless there is also supraglottic edema. However, in our experience intubation has definite disadvantages in the treatment of this disease except as a temporary relief measure until tracheotomy can be done.

In certain cases we have been successful with intubation alone in the treatment of the immediate infection but have found a complication in the form of chronic laryngeal stenosis and eventually have been required to do a tracheotomy.

One might ask why a tracheotomy is not done, then, in all cases. No one would think of operating on a patient in the face of shock but would first attempt to have the patient react. There is a condition known as respiratory shock. For instance, a patient who has been struggling for breath for several days is brought into the hospital nearly

19. Negus, V. E.: An Explanation of the Respiratory Failure Sometimes Occurring After a Successful Tracheotomy, Ann. Otol., Rhin. & Laryng. **47**:608 (Sept.) 1938.

exhausted. We have found that if we can give this patient rest for a few hours, if possible in the humidified room, and possibly an intubation, he will stand tracheotomy better than if the operation is performed immediately.

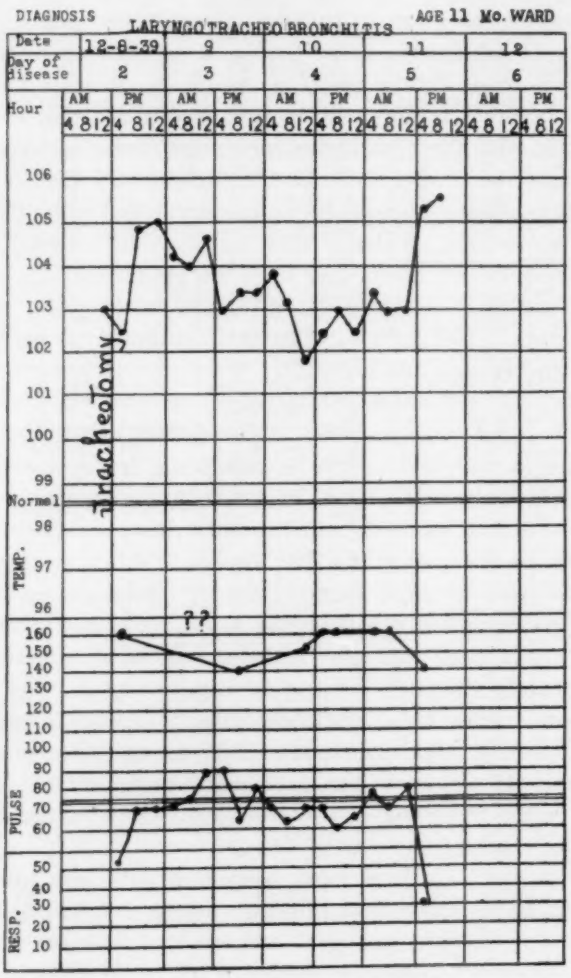

Fig. 13.—Temperature chart of 11 month old child with a fulminating acute laryngotracheobronchitis.

We believe tracheotomy to be the operative procedure of choice. It must be remembered, however, that when relief of the immediate respiratory distress is obtained, only a part of the victory is won. Simpson [20] stated that pneumothorax is a serious complication in laryngotracheo-

20. Simpson, W. L.: Pneumothorax Complicating Tracheotomy in Fulminating Laryngotracheobronchitis, Arch. Otolaryng. **26**:411 (Oct.) 1937.

bronchitis. Fortunately, we have not encountered this complication resulting from or coincident with tracheotomy. We formerly placed tracheotomized patients in a croup tent in which the air was moist but hot. During the winter of 1939-1940 we placed such patients in a humidified room in which the temperature is kept at 70 F. We found that the tracheotomized patients, as well as those on whom it was unnecessary either to intubate or to tracheotomize, did far better in this cool moist room than in a croup tent.

F. W. Davison [14] first reported the advantages of such an atmosphere before the Eastern Section of the American Laryngological, Rhinological and Otological Society, and the following information concerning the air conditioning of an oxygen tent is taken freely from a personal communication:

> I suspect that the therapy of many diseases could be improved by affording the patient certain combinations of temperature, humidity and air flow.
>
> For the treatment of acute laryngotracheobronchitis we use an oxygen tent canopy rather than an entire room for the reason that in our own room supersaturation causes the walls, etc., to drip so that paint and furnishings are ruined by moisture.

To obtain a high humidity without high temperature, we use a Walton humidifier.

When using a tent, we should supply climatic conditions which we know will be comfortable. The temperature and humidity within the canopy have more to do with the comfort of the patient than the concentrations of oxygen and carbon dioxide.

The problem is to add water vapor to the canopy more readily than it is removed by the cooling chamber. By using a humidifier which evaporates 1 pint (473 cc.) of moisture per hour, we have been able to keep the relative humidity at 95 per cent, with a temperature of 70 F., which is entirely comfortable. On the other hand, if a steam generator is connected to the canopy, conditions such as those present in the average so-called croup tent are produced, i. e., a temperature of 90 F. and a relative humidity of 95 per cent; this gives an effective temperature index of 80 degrees. These conditions are exceedingly oppressive and uncomfortable. Yet for generations croupy youngsters have been subjected to such conditions without thought of their comfort. One should not lose sight of the fact that perspiration can cause excessive loss of water. Conversely, raising the humidity while keeping the temperature low enough to prevent excessive perspiration is a means of conserving the body's supply of water. Thus, the maintaining of a high humidity, with temperature not above 70 F., is a useful method of conserving body fluids, so that an adequate volume will be available for renal function.

The postoperative care of a tracheotomized child is perhaps the most important factor in his ultimate recovery. The patient is placed in a vapor-laden room which is kept at a temperature of 70 F. He is under the constant care of a qualified nurse, especially experienced in this work. Through the tracheotomy tube, suction of the obstructing tracheal secretions can be done frequently and effectively with a rubber catheter attached to a suction machine. Solutions, such as physiologic solution of sodium chloride, a solution of sodium bicarbonate or a 1 per cent solution of monochlorophenol, may be instilled into the trachea to soften these secretions and render them more easily coughed up or aspirated by suction. If evidence of obstruction persists despite frequent aspirations by means of a catheter, the bronchoscope may be inserted through the tracheotomy wound and more effective aspiration secured. Intubation does not allow such a procedure to be carried out.

During fourteen months at the Essex County Hospital for Contagious Diseases, 16 children whose condition was diagnosed as acute laryngotracheobronchitis were tracheotomized. Five of these died, giving a mortality rate of 31 per cent. It must be expected that patients with this disease will have a stormy course after tracheotomy, and it is only under the most skilled and assiduous care of trained and conscientious personnel that the mortality rate can be lowered.

In the treatment of a staphylococcic infection the problem is to keep the bronchi as moist as possible, so as to enable the patient to expectorate the secretion. Evans [11] advocated the use of a staphylococcus phage and reported good results in his cases. The action is primarily liquefaction of the staphylococcus. The use of this agent, therefore, has some rational basis. As yet, we have not used it.

Difficulty in maintaining an adequate airway even after tracheotomy commonly results from obstruction of the trachea and bronchi with thick or inspissated secretion, and the bronchoscope must be resorted to for the removal of this. Cassidy [21] reported a case in which thirty-seven bronchoscopic aspirations were performed over a period of thirteen days. Galloway [12] advocated postural drainage as an adjunct and stated that it has proved especially valuable. With the head of the patient lowered, the bronchial lumen is kept free of secretion and plugs by the use of suction applied through a tracheotomy tube.

As to the use of sulfanilamide and sulfapyridine, these drugs were used, in conjunction with some mechanical aid, in 23 cases in our series. Our conclusion is that these drugs may help in certain cases but that they are not a specific for this disease. In any disease, rational therapy is based on a fundamental knowledge of the pathologic changes associated with that disease.

21. Cassidy, W.: Acute Laryngotracheobronchitis, Arch. Otolaryng. **29**:857 (May) 1939.

REPORT OF AUTOPSIES

AUTOPSY 1 (autopsy by Dr. C. L. Minier; sections by Dr. Lewis W. Brown).—R. T., aged 19 months, died Feb. 2, 1940. An autopsy was performed on the same day. The clinical diagnosis was acute laryngotracheobronchitis.

Gross Observations.—The body was that of a well developed and well nourished Negro boy.

Chest: The pericardium contained a moderate amount of air or gas. The heart was slightly dilated but was otherwise normal. The larynx was moderately congested and was filled with frothy fluid. The trachea was moderately congested and contained frothy fluid. The main and smaller bronchi also were congested and filled with frothy fluid. The lungs were congested and edematous. Considerable frothy fluid exuded from the cut sections. The thymus was slightly larger than normal but apparently caused no obstruction.

Abdominal Organs: All the abdominal organs appeared quite normal except the spleen, which showed hyperplasia of the malpighian corpuscles.

Microscopic Observations.—Sections from the larynx showed ulceration and necrosis of the mucous membrane lining and edema and polymorphonuclear leukocytic infiltration of the entire mucosa and submucosa. Sections from the trachea showed the epithelial layer of the mucosa to be densely infiltrated with pus. In some places there was necrosis, the purulent-necrotic exudate having a pseudomembranous appearance. The entire mucosa and submucosa of the trachea showed edema and infiltration by polymorphonuclears. The vessels of the submucosa were congested. Sections from the bronchus showed the same abnormalities as those of the trachea. Sections from the lungs showed some exudate in the bronchioles, with a zone of purulent infiltration in and around the walls of the bronchioles. The alveoli surrounding the bronchioles were filled with purulent exudate in typical lobular pneumonic distribution.

Diagnosis.—The condition was diagnosed as acute laryngotracheobronchitis and acute bronchopneumonia.

AUTOPSY 2 (Dr. C. L. Minier).—J. D., aged 14 months, died May 17, 1939. An autopsy was performed on the same day. The clinical diagnosis was tracheobronchitis and bronchopneumonia.

Gross Observations.—The body was that of a well nourished, fairly well developed white boy about 1 year of age. The skin was pale and waxlike in appearance and gave a suggestion of considerable subcutaneous edema, although there was no true pitting. It was clear and without rashes or discolorations.

Head: The head was symmetric. The anterior fontanel just admitted a finger tip. The tension was normal. The ears showed no external evidences of discharge. The pupils were equal. There was no conjunctivitis. The external nares contained a small amount of dried, crusted mucopurulent material. The lips were somewhat dry. There was no discharge from the mouth. The cranial cavity was not examined.

Chest: The chest was symmetric. There was no flaring of the ribs or beading of the costochondral junctions. The thymus was normal in size and position and did not compress the trachea; the cut surface was not abnormal. The larynx and trachea showed no evidence of obstruction to the airway. The mucous membrane lining was red and somewhat edematous and appeared moist, but there was no exudate. The bronchi were red and congested, with edematous walls, so that their lumens were slightly decreased in caliber, and they contained a considerable amount of thin reddish fluid. The pleural surfaces were smooth and glistening,

and the pleural cavities were free of fluid. At the apex of the upper lobe of the right lung was a small area of red consolidation. Here the parenchyma was firm and congested. In the upper portion of the lower lobe of the right lung was a small area of similar appearance. The left lung was rather congested and edematous but was well aerated throughout. The glands at the hili and those along the trachea and larger bronchi were slightly enlarged and appeared congested. The pericardial cavity contained no free fluid, and the epicardial surface was smooth and glistening. The chambers of the heart were not dilated. The free edges of the valves were thin, and the valves appeared competent. The endocardial surface was pale and smooth, and there was no gross scarring of the myocardium.

Abdomen: The peritoneal cavity contained no free fluid, and the peritoneal surface was smooth and glistening. The omentum was lying free and showed no congestion. The stomach contained a small amount of greenish sour-smelling material; the mucosa was pale and showed no injection. Other parts of the gastrointestinal tract were normal. The mesenteric glands were not enlarged. The liver was not enlarged, and its cut surface showed no abnormality. The gallbladder contained a small amount of viscid greenish material; the walls were thin, and the common duct was patent. The pancreas was not enlarged, and its surface showed the customary markings. The spleen was not enlarged or congested, the surface was free of adhesions and its cut surface exhibited a homogeneous red granular structure. The adrenals were not enlarged or congested and on section showed no gross evidence of hemorrhage. The kidneys were normal in size and appearance; the capsules were thin and stripped easily; the cut surface was normal. There was no dilatation of the pelves or ureters, and their mucous lining was pale. The bladder contained a small amount of clear yellowish urine. Its walls were pale and thin. The testes were in the scrotum and were not abnormal.

Microscopic Observations.—The section from the mucous membrane lining the trachea was edematous and diffusely infiltrated with polymorphonuclear leukocytes. In some areas the lining membrane had degenerated, and in others it was absent, as if there were many small ulcerations. The glands were enlarged and appeared overactive. In some areas their acinous structure had been destroyed. The sections from the lungs showed intense congestion and edema about the smaller bronchioles. The walls were swollen and diffusely infiltrated with many polymorphonuclear leukocytes. Their lumens were filled with masses of inflammatory exudate, consisting chiefly of intact and partially degenerated leukocytes and fibrinous material. The walls of the immediately adjacent alveoli were somewhat thickened and showed evidences of inflammation. In other areas there were partial collapse and atelectasis of the alveolar structures, presumably from the exudate within the terminal bronchi. No sections were taken from the areas of consolidation which were noted in the gross examination in the upper lobe and in a portion of the lower lobe of the right lung.

Diagnosis.—The condition was diagnosed as laryngotracheobronchitis with obstruction due to liquid exudate and bronchopneumonia of the upper and lower lobes of the right lung.

Autopsy 3 (Dr. Lewis W. Brown, pathologist).—R. D., aged 6 years, died Oct. 22, 1939. An autopsy was performed on the same day. The clinical diagnosis was diphtheria.

Gross Observations.—The body was that of a well developed and well nourished boy of 6 years. The skin was clear and without rashes or discolorations. There was no edema or evidence of recent loss of weight.

Head: The head was well formed. The scalp was clear. The ears showed no external discharge. The pupils were equal. There was no conjunctivitis. The external nares contained a small amount of dried mucopurulent material. The cranial cavity was not examined.

Chest: There was no beading of the costochondral junctions. There was a moderate amount of subcutaneous fat. The thymus appeared to be normal in size and position, and there was no evidence of compression of the trachea. A cut section of the thymus was normal. The pleural cavities contained no free fluid. The pleural surfaces were smooth and glistening. The parenchyma of the lungs was somewhat congested and contained a small amount of thin sanguineous fluid, but there was no gross evidence of consolidation, and the lungs were well aerated throughout. The smaller bronchioles were red and injected and contained a moderate amount of glairy bloody material. The glands at the hili and those along the large bronchi and trachea were enlarged and somewhat congested. The mucous membrane lining the larynx, trachea and large bronchi were swollen, hyperemic and edematous. Covering this surface through nearly its entire extent was a thick, somewhat adherent mucopurulent exudate, which separated easily from the underlying tissues and left no gross area of erosion. The pericardial sac contained no fluid. The epicardial surface was smooth and glistening. The chambers of the heart contained small chicken fat clots but were not dilated. The free margins of the valves were thin and appeared competent. There was no gross scarring of the myocardium.

Abdomen: There was no free fluid in the peritoneal cavity. The omentum was found to be lying free. The peritoneal surfaces appeared smooth and glistening. The liver was greatly enlarged and extended from 1 to 2 inches (2.5 to 5 cm.) below the costal margin. It was congested but soft, and its cut surface showed reddening and hyperemia of the central areas. Its large veins were filled with blood. The gallbladder was thin walled and contained a small amount of thin greenish bile. The bile duct was patent. The stomach and the intestinal tract were normal. There was no enlargement of the mesenteric glands. The pancreas was normal, and its surface exhibited the usual markings. The spleen was enlarged and was congested with blood, which exuded freely when the surface was cut. The kidneys were congested. The vessels appeared dilated, but the capsule was delicate and stripped easily, and the cut surface showed some prominence of the glomerular markings. The pelves and ureters were not dilated, and their lining was pale and smooth. The adrenals showed no abnormality, and there was no gross evidence of hemorrhage in their cut sections. The bladder contained a few ounces of pale urine. The mucosa was pale and the wall thin. The testes were in the scrotum.

Bacteriologic Findings.—Cultures of material from the trachea, bronchi and larynx were negative for the diphtheria bacillus. The predominating organism was Staphylococcus aureus haemolyticus. Diplococci, pneumococci of type III and nonhemolytic streptococci were also present.

Microscopic Observations.—No sections were made.

Diagnosis.—The condition was diagnosed as acute laryngotracheobronchitis.

Autopsy 4 (Dr. Lewis W. Brown, pathologist).—N. R., aged 11 months, died Dec. 11, 1939. An autopsy was performed on December 12. The clinical diagnosis was acute laryngotracheobronchitis.

Gross Observations.—The body was that of a well developed and well nourished white boy of 11 months. The skin was clear and without rashes or discolorations.

A recent tracheotomy wound in the lower portion of the neck was situated about 1 inch (2.5 cm.) above the jugular notch. The edges of the wound were slightly reddened and edematous, but there was no evidence of infection, nor was there any emphysema of the surrounding structures.

Head: The head was symmetric. The anterior fontanel was open, admitting a finger tip. It was not depressed. There was no external discharge from the ears. The pupils were equal. There was no conjunctivitis. Some grayish dried crusted matter lay just within the nares. The cranial cavity was not examined.

Chest: The chest was symmetric. There was no flaring of the ribs or beading of the costochondral junctions. There was a moderate amount of subcutaneous fat but no edema. The pleural cavities were free of fluid, and the surfaces were smooth. Scattered throughout all lobes of both lungs were small areas of patchy consolidation, in which the surface appeared red and congested. The smaller ramifications of the bronchial tree contained tenacious mucopurulent material, and the lining membrane was red and edematous. The other parts of the lungs contained air. The glands at the hili and along the bronchi and trachea were congested and hyperemic. The larynx, trachea and main bronchi showed swelling and hyperemia of the lining mucosa with some reduction of the caliber of their lumens. In many places a purulent plastic exudate had formed, which gave to these structures the appearance of a pseudomembranous lining. The pericardium contained 3 to 4 cc. of straw-colored fluid, but the epicardial surface glistened, and a minimal amount of fat lay beneath it. The chambers of the heart were not dilated and contained postmortem clot. The free margins of the valves were thin, the papillary muscles were not thickened and the valves appeared competent. There was no gross scarring of the myocardium. The thymus was not enlarged, and its cut surface was normal.

Abdomen: The peritoneal surface was smooth and glistening, the omentum was free and the individual viscera occupied their normal positions. The liver was not enlarged or engorged and on section showed the customary markings. The gallbladder was thin walled and contained 8 to 10 cc. of greenish bile. The duct was patent. The stomach and intestinal tract were normal. There was no enlargement of the mesenteric glands. The pancreas was normal in size, and sections showed no abnormality. The kidneys were buried in perirenal fat and were not enlarged. The capsules stripped easily, and the cortical markings were not distorted. The pelves and ureters were not dilated. The lining membranes were pale and smooth and showed no evidence of inflammation. The adrenals were not enlarged or hyperemic, and there was no gross evidence of hemorrhage. The testes were within the scrotum.

Microscopic Observations.—Sections from the lungs showed the small bronchi to be filled with purulent exudate and the walls to be diffusely infiltrated with polymorphonuclear leukocytes. In areas the lining epithelium had partially degenerated, and in others it was entirely missing. These structures were edematous, and the blood vessels were congested. The alveoli surrounding these bronchi were distended with purulent exudate, consisting chiefly of myriads of polymorphonuclear leukocytes and of fibrinous material, together with degenerated epithelial cells. The alveolar walls were edematous and thickened, and in some areas there was partial collapse of the alveolar structure.

Diagnosis.—The condition was diagnosed as acute laryngotracheobronchitis and bronchopneumonia.

Autopsy 5 (Dr. Lewis W. Brown, pathologist).—E. H., aged 2 years, died March 2, 1937. An autopsy was performed on the same day. The clinical diagnosis was foreign body in the trachea. A tracheotomy had been performed.

Gross Observations.—The body was that of a well developed and well nourished girl of 2 years. The skin was clear, with no rashes or discolorations. There was a recent tracheotomy wound in the neck, about 2 inches (5 cm.) in length and lying about 1 inch (2.5 cm.) above the manubrium sterni. The edges were somewhat red and edematous, but there was no gross evidence of infection, and the underlying structures appeared to be free of infection.

Head: The head was well formed. The anterior fontanel was closed. There was no gross discharge from the aural canals. The pupils were somewhat constricted but equal in size. The conjunctivas were not injected. A small amount of dried greenish material was noted in the external nares. The lips were dried and moderately fissured. The teeth were in good condition. The cranial cavity was not examined.

Thorax: The chest was well formed. There was no beading or flaring of the ribs. The pleural cavities contained a very small amount of clear straw-colored fluid (10 to 15 cc.). There were no adhesions, and the pleural surfaces were smooth and glistening. The lungs appeared edematous and congested, and on section contained a moderate amount of thin watery sanguineous exudate. There was no evidence of pneumonic consolidation. The lymph glands about the hilus and along the bronchi and trachea were congested and slightly enlarged but not discolored. The larynx and trachea were edematous and hyperemic and appeared acutely inflamed. The vocal cords were red and showed very slight thickening, but the airway appeared adequate. Below this region a greenish plastic exudate completely lined the lowermost portion of the larynx. Beneath this the mucosal lining was red and hyperemic but apparently intact, and there were no gross erosions. Lower, the trachea and bronchi showed a pseudomembranous inflammation of the mucous membrane. Thick tenacious material of a dirty gray green color was moderately adherent to the mucosa. When this was removed, the underlying structures were intensely red, congested and edematous, and in the smaller ramifications of the bronchial tree was an occasional area which appeared to be nearly obstructed, but there were no areas of true atelectasis surrounding them. The mediastinal tissues were edematous and showed evidence of emphysema, which apparently originated at the site of the tracheotomy wound. The pericardial sac was free and contained no fluid. The heart was not enlarged. The epicardium was smooth, and the myocardium showed no evidence of fibrosis. The ventricular chambers contained a small amount of postmortem clot. The valves were thin and delicate, the papillary muscles were not thickened and the valves appeared competent.

Abdomen: The peritoneal surface was smooth and glistening. There was no free fluid. The omentum was free and clear. The abdominal viscera occupied their normal positions. The liver was not enlarged or congested. On section it exhibited the usual markings. The gallbladder contained a small amount of viscid green bile. The duct was patent, and the walls were not thickened. The mesenteric glands were not enlarged, and the stomach and intestinal tract were normal. The pancreas showed no evidence of inflammation, and its cut surface revealed the customary lobulations. The spleen was not enlarged but was minimally congested. Its capsule was smooth and free of adhesions, and transection showed a red granular surface. The adrenals were not enlarged or congested, and gross hemorrhage was not noted within their structure. The kidneys were of normal size, their capsules stripped easily and on gross section the cortex and medullary portions appeared normal. The pelves and ureters were not dilated. Their lining membrane was pale and smooth and showed no inflammatory process. The bladder was pale

and thin walled and contained a few ounces of pale yellow urine. The uterus, tubes and ovaries were normal.

Bacteriologic Findings.—Cultures of material from the larynx and trachea were negative for diphtheria bacilli. Both showed staphylococci, streptococci and gram-negative (otherwise unidentified) bacilli.

Diagnosis.—Acute laryngotracheobronchitis.

Autopsy 6 (Dr. Lewis W. Brown, pathologist).—T. B., aged 6 months, died Nov. 22, 1936. An autopsy was performed on Nov. 23. The clinical diagnosis was diphtheria (?).

Gross Observations.—The body was that of a moderately well developed, fairly well nourished white boy of about 6 months. The skin was clear, with no evident rashes or discolorations.

Head: The head was well formed. The anterior fontanel was open and slightly depressed. It measured about 1 by 1.5 cm.. There was no gross discharge from the ears. The nose was moderately crusted, and there was a small amount of purulent material within the external nares. The eyes were clear. There was no conjunctivitis. The cranial cavity was not examined.

Chest: The chest was symmetric. There was no beading or flaring of the ribs. The thymus appeared to be enlarged, particularly in the anterior-posterior diameter, and compressed the trachea posteriorly. It weighed 11 Gm. and on section appeared normal. The posterior pharyngeal wall was moderately injected and somewhat edematous. The larynx, while slightly reddened, did not appear to be otherwise abnormal. The vocal cords were smooth and not swollen; there appeared to be no obstruction of the airway at this point. Just below the larynx (½ inch [1.27 cm.]) was an area of marked constriction of the trachea, so that the lumen was definitely decreased in caliber. At this point the mucosal lining was reddened and slightly thickened. The larger bronchi were not remarkable and contained only a small amount of mucoid and mucopurulent material. The smaller bronchi and the terminal bronchioles were diffusely reddened. The mucosal lining was injected and edematous and partially covered by moderate amounts of stringy, somewhat adherent mucopurulent exudate. The lungs were well aerated throughout, and there were no areas of consolidation or scarring. The perihilar glands and those along the trachea and bronchi were not enlarged or discolored. The pericardial sac was free and contained no free fluid or adhesions. The pericardial surface was smooth and glistening throughout. The heart contained a small chicken fat clot; there was no dilatation of the ventricular chambers. The myocardium showed no gross scarring and the endocardial surface was smooth. The free margins of the valves were thin and appeared competent. The aorta was not remarkable.

Abdomen: The peritoneal surface was smooth and glistening. The individual viscera occupied their usual positions. There was no enlargement of the mesenteric glands. The omentum lay free within the abdomen; it was not thickened or injected. The liver was not enlarged or congested, and on section it exhibited the usual markings. The gallbladder contained a small amount of greenish bile; the walls were thin and the duct was patent. The stomach contained a small amount of sour-smelling stringy fluid. The intestinal tract appeared normal throughout its entire length. The spleen was not enlarged or congested. Its cut surface was granular. A small accessory spleen measuring 1 by 1 cm. was present. The pancreas was normal. Gross section showed the usual markings. The adrenals were not enlarged or congested. Transection showed no gross evidence of hemorrhage, and the medullary and cortical portions were normal. The kidneys were

of normal size and were not congested. The capsule stripped with ease. The pelves and ureter were not dilated and showed no evidence of infection. The bladder contained a small amount of clear yellow urine. Its wall was pale and smooth. The testes were in the scrotum. The extremities exhibited no gross abnormalities.

Diagnosis.—The condition was diagnosed as congenital atresia of the trachea and acute pharyngotracheobronchitis.

AUTOPSY 7 (Dr. Lewis W. Brown, pathologist).—F. P., aged 21 months, died Dec. 20, 1934. An autopsy was performed on December 21.

Gross Observations.—The body was that of a well developed boy aged about 1½ years, showing moderate evidence of dehydration. The skin was clear, and there were no rashes or discolorations. The clinical diagnosis was laryngotracheitis.

Chest: The pleural cavities contained no free fluid. No adhesions were noted, and the pleural surfaces were smooth and glistening. Both lungs showed diffuse areas of patchy consolidation, the cut surface being red and firm, with an appearance of acute congestion. The posterior portion of the lower lobe of the right lung was entirely consolidated and partially atelectatic. The cut surface was dark red, hard and somewhat depressed and was more homogeneous than the surrounding tissue. The glands at the hilus, about the bifurcation of the trachea and above this point were all enlarged and congested. The mucous membrane lining the larynx and trachea was hyperemic and edematous and in several small areas was definitely hemorrhagic. At the junction of the lower end of the larynx and the trachea was a definite constriction. At this point some erosion of the mucosa and underlying structures, possibly due to attempts at intubation, was noted. There were marked swelling and edema of all the structures, so that the tracheal lumen was encroached on and moderately decreased in diameter. The pericardial surface was smooth and glistening. The pericardial cavity contained about 5 cc. of clear serous fluid. The heart appeared grossly normal. The valves were free and appeared to be functionally adequate, and no scars were seen in the myocardium. The right chamber of the heart was dilated and filled with chicken fat clot. The thymus weighed 14.7 Gm. and was normal on section.

Head: The head was symmetric. The pupils were somewhat constricted but equal. The conjunctivas was clear. The ears showed no external discharge. The nares were crusted and contained a small amount of purulent material. The cranial cavity was not examined.

Abdomen: There was no free peritoneal fluid. The peritoneal surface was smooth and glistening throughout. The individual viscera appeared to occupy their normal positions. The liver was somewhat distended and slightly congested but on section presented the customary markings. The gallbladder contained a small amount of greenish bile. Its walls were not thickened. The stomach and intestines were normal. There was no enlargement of the mesenteric glands. The kidneys were slightly congested, but the capsules stripped easily and on section appeared normal. The pelves and ureters were smooth and showed no gross evidence of inflammation. The bladder was normal and contained a small amount of clear yellow urine. Both testes were within the scrotum. The adrenals were in their normal position and showed no gross abnormality.

Microscopic Observations.—(The lungs only were examined.) Sections from the lung showed large and small areas of purulent bronchopneumonia. The alveolar spaces were stuffed for the most part with collections of polymorphonuclear leuko-

cytes, and in some areas there were small patches of hemorrhage. The alveolar walls were edematous and thinned out, but no ruptured alveoli were seen. The terminal bronchioles were filled with masses of inflammatory exudate, and in many areas the mucosal lining was missing. The underlying structures were diffusely infiltrated with numerous polymorphonuclear leukocytes, and some early fibroblastic reaction was noted. A section from the right lobe showed collapsed alveoli, with a few areas containing gross hemorrhages. The blood vessels were distended with blood, and the smaller branches of the bronchial tree contained inflammatory exudate. In other areas the bronchi were relatively clear and the lining appeared to be intact, though even in these areas a slight infiltration of the bronchial walls with inflammatory cells was noted.

Diagnosis.—The diagnosis was acute laryngotracheobronchitis and bilateral bronchopneumonia.

SUMMARY AND CONCLUSIONS

An analysis of 62 cases of acute laryngotracheobronchitis, with reports of autopsies in 8 cases is submitted.

Acute laryngotracheobronchitis in young children is an important problem both for the general practitioner and for the pediatrician.

Proper handling of this disease requires early recognition. When signs of obstructive dyspnea appear, valuable time should not be lost in treating the condition with diphtheria antitoxin.

When acute laryngotracheobronchitis is suspected, the patient should be transferred immediately to a hospital where direct laryngoscopy and bronchoscopy can be carried out at once and where more active treatment, such as intubation and tracheotomy, may be done at a moment's notice, with an especially trained personnel always immediately available.

If time permits, roentgen study should be made prior to operation; otherwise it should be done immediately after tracheotomy.

Intubation should be done only for immediate relief; this should be followed by tracheotomy.

Patients should be placed in a room in which the air is supersaturated with moisture and maintained at a temperature of 70 F. at all times. This temperature and high humidity provide a more comfortable environment for the patient than the old-fashioned hot croup tent.

By the use of a mechanical humidifier the humidity in a tent can be raised to 95 per cent when the room temperature is 70 F.

The temperature and humidity of an oxygen tent have more effect on the comfort of the average patient than does the concentration of oxygen.

If the temperature of the air is not above 70 F., loss of water from the body can be decreased by increasing the humidity.

Tracheotomy is the operation par excellence.

Members of the staff of the Essex County Hospital for Contagious Diseases assisted in the preparation of this paper. Dr. Lewis W. Brown prepared the reports of autopsies.

CHEMOTHERAPY

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Chemotherapy will aid the otorhinolaryngologist in contributing to the health and happiness of patients with infections of the middle ear, mastoid, nasal accessory sinuses, nasopharynx and larynx. It will possibly save the lives of patients with complications such as osteomyelitis of the cranial bones, meningitis, lateral sinus thrombosis and brain abscess.

Most of the suggestions given here for the chemotherapy of infections are based on experiments made by the marrow culture method.[1] This method permits controlled quantitative study in the presence of living human cells of the relative effectiveness of different therapeutic agents against many types of micro-organisms and comparison with a control which is untreated. It has great advantages over clinical investigation and animal experimentation.[2] The observations made on chemotherapy by this method have been confirmed by subsequent clinical studies except in those cases which are too recent for clinical studies to have been completed. Other suggestions given here are based on clinical experience with the drugs concerned and on the extensive literature which has appeared in the field of chemotherapy.[3] Time and space will not permit presentation of the evidence for the principles of therapy which are to be given, but this evidence will be found in the references.[4]

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(Footnote continued on next page)

GENERAL PRINCIPLES

The following suggestions are presented in the form of general principles, as they apply to chemotherapy in all fields of medicine, but the specific examples are chosen from the field of otorhinolaryngology.

The first problem to be decided is: Does a particular patient need chemotherapy? This may usually be decided by weighing the risk of the therapy against the risk of the disease, taking into consideration the benefits which chemotherapy will offer the patient. From the data available, it appears that even with the best care to prevent toxic effects the mortality with any of the chemotherapeutic agents with which this paper is concerned (sulfanilamide and its derivatives and neoarsphenamine) is about 1 per cent; obviously, one will not want to use chemotherapy in conditions in which there is little or no mortality, such as simple nonspecific pharyngitis or mild laryngitis. Since none of the drugs now available has proved to be effective against virus diseases and since influenza is a virus disease, there is no indication for the use of any of these agents in the type of influenza epidemic being encountered at the time of writing. However, in an epidemic of influenza such as that of 1918, chemotherapy with one of these agents might be well worth while as a prophylactic measure against the complication of beta hemolytic streptococcus pneumonia, which led to a high mortality in that epidemic. Since mortality from the use of these drugs can be

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prevented only if a leukocyte count, a hemoglobin determination and a physical examination are made daily, the drugs should not be administered unless the patient is sick enough to justify this measure and a daily visit by the attending physician. If it is remembered that the mortality from the use of these drugs is greater than that from the use of general anesthetics, one will rarely make the mistake of prescribing the drugs for infections that are too minor to justify their use.

The next point to determine is which drug to use. This depends on the organism responsible for the infection. Thus, in any type of infection, smears and cultures are much more important now than formerly. The best method of obtaining material for smears and cultures is aspiration with a sterile syringe and needle. If possible, at least 1 cc. of purulent material should be procured from the site of infection. The organisms most frequently encountered [5] in infections with which otorhinolaryngologists have to deal are the pneumococcus, the beta hemolytic streptococcus, the alpha hemolytic streptococcus (Streptococcus viridans), the gamma streptococcus, Staphylococcus aureus, the influenza bacillus and the diphtheria bacillus. Many other organisms may be encountered occasionally. The effectiveness of many of the derivatives of sulfanilamide and of many arsenicals against each of these organisms except the influenza bacillus and the diphtheria bacillus has been studied by the marrow culture technic. The study has shown that sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) is equal to or superior to sulfanilamide against the beta hemolytic streptococcus and equal to or somewhat superior to sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) against the pneumococcus. Sulfathiazole is far superior to either sulfanilamide or sulfapyridine in staphylococcic infections, is much superior to sulfanilamide against the alpha hemolytic streptococcus (Str. viridans) and is effective against many organisms, including among others the typhoid bacillus, the paratyphoid bacillus, the colon bacillus, Staph. aureus and Staphylococcus albus, against which neither sulfanilamide nor sulfapyridine is very effective. Furthermore, since many infections with which the otorhinolaryngologist deals are mixed infections, in my opinion sulfathiazole is the drug of choice among the derivatives of sulfanilamide so far studied. However, neoarsphenamine is superior to sulfathiazole against many strains of Str. viridans, and sulfathiazole alone is entirely ineffective against some of these strains against which neoarsphenamine is very effective. Against staphylococci both neoarsphenamine and sulfathiazole are very effective, so

5. Ashley, B. J., and Frick, W. V.: A Bacteriologic and Cytologic Study of the Maxillary Antrum in Children, with a Clinical Study of Eighty-Three Cases, Ann. Otol., Rhin. & Laryng. **39**:605-630 (June) 1930. Kistner, F. B.: Chronic Nonpurulent Sinusitis and Its Clinical Significance, ibid. **38**:795-804 (Sept.) 1929.

that administration of both drugs is recommended in the more serious staphylococcic infections. The principle, therefore, is to use the drug which is most effective against the particular infecting organism. At present, except in the more serious staphylococcic and Str. viridans infections, this drug is sulfathiazole. In infections of those types neoarsphenamine, either in conjunction with or instead of sulfathiazole, may be preferable.

The next question to decide is: When should the administration of the drug be started and how should it be carried out? All our experimental data and that of others tend to show that the fewer the organisms present the more effective are the drugs. Therefore, in acute infections it is important to begin therapy at the earliest possible moment, i. e., as soon as it is decided that the administration of drugs is indicated. If the infection is severe, this may be done as soon as material for smears and cultures has been taken, even before the organism has been identified. It is important also to take blood for a leukocyte count and a hemoglobin determination before starting the administration of drugs, since if the first count is taken only after the drug has been started it is often impossible to tell whether leukopenia or anemia is due to the drug or to the disease. If the abnormality of the blood is due to the drug, the only way to save the life of the patient is to stop administration of the drug immediately. If it is due to the disease, the only way to save the patient's life may be to continue giving the drug in maximum dosage; hence, the importance of a preliminary routine hematologic examination is clear. It has been determined by studies carried out with marrow culture that the ideal blood level of sulfathiazole against any of the organisms named is 5 to 8 mg. per hundred cubic centimeters. Clinically, blood level determinations are essential to the accurate maintenance of the blood level, because of wide variations in the absorption of the drug. It is essential that the blood level of the drug be maintained over a long period, for marrow cultures have shown that the failure to maintain an adequate concentration for forty-eight to seventy-two hours has prevented complete sterility and often a much longer time has been necessary to destroy the organisms, although their number may have been reduced to only 1 to 10 per cubic centimeter. Such a small number of organisms does not cause clinical symptoms but is sufficient to cause a recrudescence of the infection if administration of the drug is stopped. It is therefore important to give the drug at sufficiently frequent intervals to obtain a uniform blood level and, if toxic effects do not prevent, to continue therapy for three to four days and preferably a week after all symptoms of the disease are over and cultures are negative. For adults the initial dose of sulfathiazole should be 2 to 4 Gm., with 1 to 2 Gm. every four hours day and night thereafter. The blood level should

be checked each morning, and the dose should be changed as necessary to keep it between 5 and 8 mg. per hundred cubic centimeters. If the infecting organism is Staphylococcus or Str. viridans and the patient is seriously sick, as with a bacteremia or meningitis, neoarsphenamine should be given according to the following schedule.

Give intravenously a unit dose of 0.8 mg. of freshly prepared neoarsphenamine per pound (0.5 Kg.) of body weight every four hours for four doses the first day and every eight hours for three doses each subsequent day. For acute staphylococcic infections, continue for ten days. For subacute bacterial endocarditis or other Str. viridans infections, continue for six days. If the temperature is then normal, alternate three to six day rest periods with three day courses of therapy and begin each new course with the first day regimen. If the temperature is unaffected, discontinue the administration of neoarsphenamine and give sulfathiazole every four hours day and night in doses sufficient to maintain a blood level of 5 to 8 mg. per hundred cubic centimeters. If the temperature is lowered but not normal, give both neoarsphenamine and sulfathiazole. If there is no evidence of serious toxic effects, continue therapy for at least sixty days.

This method of administration of neoarsphenamine has been shown to maintain blood levels at 100 to 130 micrograms of arsenic per hundred cubic centimeters, corresponding to a concentration of 1 : 150,000, which was shown by our marrow culture studies to be the ideal cencentration.[6] Twice this concentration killed the cells in the marrow cultures, and half this concentration failed to kill the organisms. It is essential, therefore, that the schedule for dosage be followed accurately. In case of serious staphylococcic infection, both neoarsphenamine and sulfathiazole should be given. In cases of meningitis, sulfapyridine may prove to be the drug of choice, because there is some evidence that sulfathiazole does not enter the spinal fluid as readily as sulfapyridine. However, since there is a difference of only five points in the molecular weight, further work must certainly be done before this can be accepted as definitely established. In cases of serious pneumococcic infections, type specific antiserum should be given as well as sulfathiazole or sulfapyridine.

If local collections of pus are present, adequate drainage should be instituted at once and the cavity should be filled with sulfathiazole crystals, which are now available, at least for investigational purposes, in 2 Gm. sterile ampules. In wounds or cavities from mastoidectomies, operations for osteomyelitis, tooth extractions or operations in infected areas such as the nose, mouth and throat, the powdered crystals may be poured or blown in. Some locations suggest that a heavy suspension in sterile saline solution is preferable; others, such as an antrum which has been drained from below, that a heavy suspension in a viscid water-soluble medium would be better, since such a medium would be less

6. Osgood, Joski and Brownlee.[4g] Osgood.[4b,1]

likely to run out. Meacham and I [1] are investigating the use of the water-soluble lubricating jellies as suspending mediums as well as that of other preparations having this general consistency. The results of these studies will be published elsewhere. Possibly some members of this society who have equipment for carefully controlled studies of such a preparation would like to aid us in these studies. However, it must be kept in mind that this investigation is still in the experimental stage. The reason for the local use of sterile powdered crystalline sulfathiazole is that none of the drugs in the concentrations obtainable in the blood stream is effective against large numbers of organisms. This has been shown in marrow cultures. Collections of pus, such as may occur in acute sinusitis, acute otitis media and acute mastoiditis, nearly always contain large numbers of organisms. By opening and draining such a collection of pus and gently washing the cavity with a sterile saline solution saturated with sulfathiazole one greatly reduces the number of organisms. The local application of powdered sulfathiazole then raises the local concentration of the drug to ten times that obtainable by giving the drug by mouth, and the relative insolubility of the drug prevents too rapid absorption and permits use of a much larger quantity (2 to 30 Gm.) than would be possible if the drug were immediately dissolved and absorbed. However, the sulfathiazole is slowly dissolved and absorbed, so that more must be added from time to time as it disappears. Our clinical experience with this method of using the drug in conjunction with a sufficient oral dose to maintain a blood level of 5 to 8 mg. has been extremely satisfactory. In patients with meningitis, it is essential to drain the infected sinus or area of osteomyelitis at the earliest possible moment, even if previous experience with such patients leads one to believe the outlook is hopeless. Dr. J. G. M. Bullowa, of New York, in collaboration with whom most of my work on pneumococcic infections has been done, found that there was a much lower mortality in a group of patients with pneumococcic meningitis who were treated with sulfapyridine, antiserum and the drainage of a local collection of pus than in a group in which similar treatment except for the drainage was given. It seems certain that the use of sulfathiazole crystals in drained areas such as these will lower the mortality still further. The maintenance of an adequate blood level lessens the danger of introducing organisms into the blood stream, which was formerly a major reason for postponing operation in cases of infection. Of course, it is still important to use the utmost gentleness in draining infected areas, to avoid as far as possible the destruction of the natural barriers which prevent infection of the blood stream. In chronic infections of

7. Osgood, E. E., and Meacham, P. L.: Local Use of Powdered Crystalline Sulfathiazole in Viscid Aqueous Suspension in Dental Surgery, to be published.

sufficient severity to justify the use of the chemotherapeutic agents named here, it would seem desirable to use them locally in the manner described as well as by mouth, although even with this method of administration it must be remembered that the drugs may not penetrate to the organisms embedded in the tissues and that there is not as yet sufficient clinical evidence to determine how effective the drugs will be.

When the blood levels recommended are maintained, toxic effects from sulfathiazole [8] and neoarsphenamine are due to idiosyncrasy rather than to the size of the dose; therefore, every patient should be observed daily for toxic manifestations. The most serious toxic reactions associated with either drug are agranulocytosis, severe anemia, impaired renal function and skin rashes. However, either drug may produce fever, jaundice or polyneuritis. The use of sulfathiazole may lead to severe conjunctivitis or to anuria from the deposition of crystalline sulfathiazole, not acetyl sulfathiazole, in the urinary tract. In rare instances the administration of neoarsphenamine leads to hemorrhagic encephalitis, hepatitis or aplastic anemia. A drop in the leukocyte count below 4,000 per cubic centimeter, a drop in hemoglobin of more than 20 per cent in one day or the development of a sudden decrease in the volume of urine, when any of these is due to the drug and not to the disease, is an imperative indication for the immediate discontinuance of chemotherapy. With other toxic manifestations the seriousness of the disease must be weighed against the seriousness of the toxic manifestations, although a skin rash associated with the administration of neoarsphenamine usually indicates the withdrawal of the drug unless the patient's life is in immediate danger from the disease. Since sulfathiazole may cause some mental confusion and muscular incoordination, it is important that patients who are receiving this drug be instructed not to drive cars or to engage in other activities in which some muscular incoordination or mental confusion might endanger the lives of themselves and others. Vomiting is a frequent complication of sulfathiazole therapy and is not an indication for stopping administration of the drug unless the infection is the mildest for which the drug is indicated. Administration by rectum in double the usual doses will usually succeed in maintaining adequate blood levels. If not, sodium sulfathiazole may be injected intravenously, but great care must be taken in entering the vein, as this drug is extremely caustic; and under no circumstances should it be given by any other route. To unconscious patients it is simpler to give sulfathiazole in suspension in water or other liquid by a stomach tube or by rectum than to give it intravenously. Fever may

8. Long, P. H.; Haviland, J. W.; Edwards, L. B., and Bliss, E. A.: The Toxic Manifestations of Sulfanilamide and Its Derivatives, with Reference to Their Importance in the Course of Therapy, J. A. M. A. **115**:364-368 (Aug. 3) 1940.

be produced by either sulfathiazole or sodium sulfathiazole; so if fever persists after the patient has seemed to be clinically well and cultures have been negative for a week, the drugs may be cautiously discontinued, with watchfulness for the effect on the temperature curve. If the temperature becomes normal and on readministration of the drug there is another rise, it is probable that the fever was due to the action of the drug itself. In any event, for the few days following discontinuance of the drug the patient should be observed closely for evidence of a recrudescence of the disease.

SUMMARY

Evidence from quantitative controlled studies carried out with cultures of human marrow indicates that at present the chemotherapeutic agent of choice against the majority of infections is sulfathiazole. Against the most severe staphylococcic or Str. viridans infections the drug of choice is neoarsphenamine, either alone or in combination with sulfathiazole. To determine whether the drugs are indicated or not, it is important to weigh the risk of the therapy against the risk of the disease and to know that the organism producing the disease is susceptible to the action of the drug being used. After it has been determined that the severity of the infection warrants the use of one of these drugs, administration should be begun at once. The drug (sulfathiazole) should be administered every four hours, day and night, preferably by mouth, in quantities sufficient to maintain a blood level of 5 to 8 mg. and sometimes 10 mg. per hundred cubic centimeters. In serious pneumococcic infections type-specific antipneumococcus serum should be used in connection with sulfathiazole or sulfapyridine. Local collections of pus should be drained at once and crystalline sulfathiazole in powdered form introduced either as a dry pack for the wound or in heavy suspension in saline solution or some viscid water-soluble suspending medium which is nonirritating and nontoxic. Sterile powdered crystalline sulfathiazole seems to be of great value in the prophylactic treatment of postoperative wounds in infected areas. In serious staphylococcic or Str. viridans infections neoarsphenamine should be used in the dosage outlined. This will maintain a uniform blood level of 100 to 130 micrograms of arsenic per hundred cubic centimeters, corresponding to a concentration in the blood of 1 : 150,000. Unless there are toxic reactions or danger of them, administration of these drugs should not be discontinued until about a week has elapsed after the temperature has become normal and the cultures are negative. If avoidable mortality is to be prevented, careful watch must be kept for toxic manifestations due to idiosyncrasy when either sulfathiazole or neoarsphenamine is used. Chemotherapy offers promise of reducing greatly the mortality and morbidity from infection.

CHANGES IN VESTIBULAR SENSITIVITY IN MÉNIÈRE'S SYNDROME AND THEIR SIGNIFICANCE

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It has been common ground with all observers that tests of vestibular function in cases of Ménière's syndrome are of no value because of the conflicting results. One patient will show hyperfunction and another will show hypofunction in the affected ear, while a third will show no difference between the two sides. The fact of these variations in function between individual patients cannot be gainsaid, but I submit that the deduction made from this fact, that vestibular tests are valueless in this condition, is fallacious. That function varies is surely evidence enough that the results mean something if only sufficient information is at hand to assess them. This paper will, I hope, show that such information is now available.

THE QUANTITATIVE COLD CALORIC TEST

In a recent communication [1] a method of performing the cold caloric test was described which conforms to clinical requirements in that it is not too elaborate, and therefore time consuming, and is yet quantitative and accurate. By its means, a standard norm of vestibular function has been established, which depends on the relation between the time of onset of nystagmus and its time of duration. This relation in the normal subject is as 1 is to 4. It is diminished in the presence of hypofunction —1 : 2, for instance, or even 2 : 1. It is increased in the presence of hyperfunction, even to 1 : 7.

By the use of this method consistently in cases of Ménière's disease, it has been found, in conformity with other observers, that there was an apparent inconsistency in the results. Even in the same patient from examination to examination differences have been observed—on one occasion diminished function, on another equal function. Change from diminished to increased function or vice versa has not, however, been observed. Nor have the extreme changes been observed which were

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1. Atkinson, M.: A Simple Quantitative Method of Testing Vestibular Function, Arch. Otolaryng. **30**:916 (Dec.) 1939.

described by Crowe,[2] who said: "Some patients with Ménière's disease have total absence of response to the caloric test at one examination while at a later test with the same technique a characteristic nystagmus response is obtained." Nevertheless, all observers agree that changes in degree of sensitivity of the affected labyrinth do occur, and without obvious rhyme or reason. It was not until it was realized that there are two varieties of Ménière's disease, the same syndrome being produced by two different mechanisms, that order began to come out of chaos.

THE MECHANISM OF MÉNIÈRE'S SYNDROME

This subject has been considered in detail elsewhere.[3] Briefly, it appears that Ménière's syndrome can be produced by either of two mechanisms, the one allergic and the other vasospastic. These can be differentiated by means of the histamine skin test, the allergic group giving a positive reaction and the vasospastic group a negative.

THE RELATION OF VESTIBULAR FUNCTION TO THE HISTAMINE TEST

When the vestibular function came to be correlated with the cutaneous reaction to histamine, it was found that in the allergic group vestibular function was either diminished or unchanged, while in the vasospastic group it was almost invariably increased.

Of 22 patients with the Ménière syndrome on whom a histamine skin test was done, 8 reacted positively and have been treated with histamine phosphate for desensitization with success, the therapeutic response demonstrating the correctness of the diagnosis.[3] Of these 8, 4 showed hypofunction of the affected labyrinth between attacks; in 3 function was normal; in 1 a quantitative test was impossible owing to a radical mastoid operation having been performed after a mistaken diagnosis. *In none was hyperfunction found.* Moreover, this group comprised all the patients with hypofunction except 2. These, with the 3 showing normal function, will be discussed later.

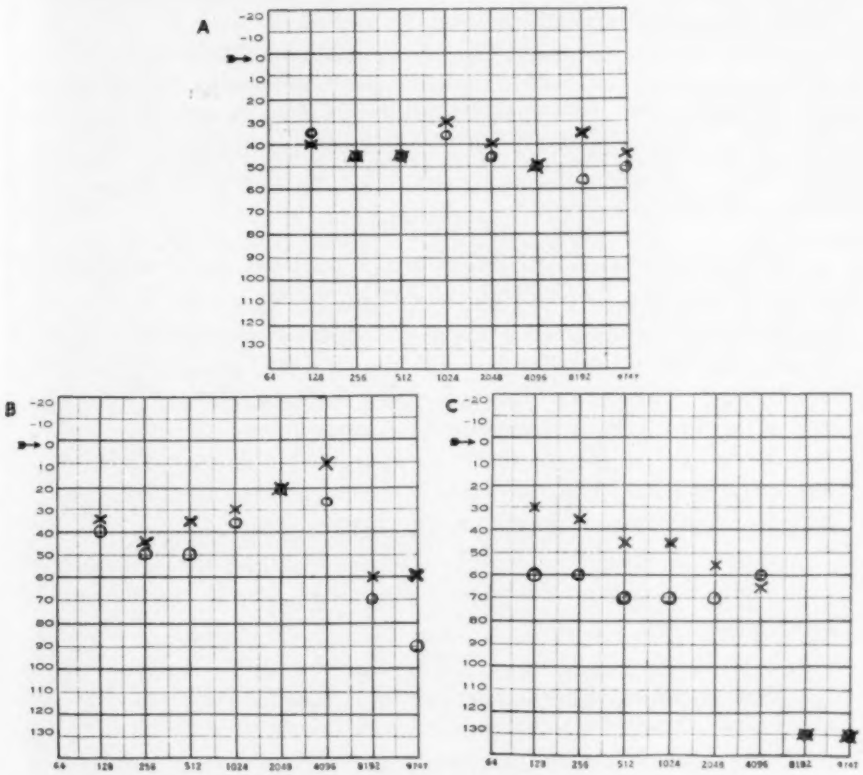
EXAMPLE 1.—A woman of 32 had severe attacks of vertigo, the first two months before she was examined, with intermediate and indeterminate dizziness. Diminished hearing and tinnitus on the right side had arisen coincidentally. There was a history of sneezing and spasmodic rhinorrhea. She gave a markedly positive cutaneous reaction to histamine. Labyrinthine function on the unaffected side was normal, with onset of nystagmus in twenty-five seconds and duration one hundred seconds ($25/100 = 1:4$). On the affected side the onset was slower, thirty-five seconds, and the duration shorter, sixty seconds ($35/60 = 1:2$, or rather less).

2. Crowe, S. J.: Ménière's Disease: A Study Based on Examination Made Before and After an Intracranial Division of the Vestibular Nerve, Medicine **17**:1 (Feb.) 1938.

3. Atkinson, M.: Observations on the Etiology and Treatment of Ménière's Syndrome, J. A. M. A. **116**:1753 (April 19) 1941.

Thus there was marked hypofunction of the vestibule on the affected side, associated with a positive reaction to histamine test. Histamine desensitization was successful.

On the other hand, 14 patients reacted negatively to the histamine skin test and were therefore placed in the second, or vasoconstrictor, group. These were treated with considerable success on lines directed toward overcoming the vasospasm.[3] Of this group, all except 2, no



Audiograms of: *A*, a woman aged 26, with vertigo of six months' duration; *B*, a woman aged 42, with symptoms of twenty years' duration; *C*, a woman aged 35, who had had attacks of Ménière's disease for nine years.

matter what their age, showed hyperfunction of the vestibule on the side of the affected ear, the 2 exceptions being those already mentioned as the only patients with hypofunction who did not come into the vasodilator group. Every patient in whom hyperfunction was present fell into this group of persons with a negative reaction to the histamine test, the postulated vasoconstrictor group.

EXAMPLE 2.—The increase in sensitivity, though definite, is not usually extreme. Thus a man aged 29, whose attacks had been present for three years, with deafness and tinnitus on the right side, showed labyrinth function on the left 30/105 and on the right 23/140 (1 : 3½ and 1 : 6 respectively).

EXAMPLE 3.—This hyperfunction is present also in elderly persons. A man aged 64, with a blood pressure of 150 systolic and 70 diastolic, had suffered eight typical attacks of Ménière's disease in one year, with bilateral deafness and tinnitus, more marked and of longer duration (six years) on the left side. Vestibular function in the less deaf right ear was 25/115; that in the more deaf left ear, 20/120. There was a degree of bilateral hypersensitivity, more severe on the left and more affected side (right, 1 : 4½; left, 1 : 6).

EXAMPLE 4.—The discrepancy can be considerable even in early stages of the disease. A woman aged 54 had suffered many attacks over fourteen months. She had deafness and tinnitus on the left side, which had appeared two weeks before her first attack. Vestibular function in the unaffected right ear was 30/110, and that in the affected left ear, 20/110 (right, 1 : 4; left, 1 : 5½).

AUDIOGRAMS

As an additional aid to differentiation in those cases in which the results of the tests are doubtful, the audiogram has a place to fill.

The characteristic audiogram of the vasodilator group is that of a usually bilateral conductive deafness, the straight line across the chart, which shows lowering of hearing for all tones (chart, *A*). This applied to 7 of the 8 patients in this group. The audiogram of the eighth, whose symptoms were of twenty years' duration, was that characteristic of a late stage of conductive deafness—a loss of hearing for both low and high tones, with a peak in the middle register (chart, *B*).

In the vasoconstrictor group every patient had the characteristic audiogram of perceptive deafness—a precipitous drop in hearing for high tones (chart, *C*).

COMMENT

There is a general agreement nowadays that the labyrinth is the site of the disturbance in Ménière attacks of classic type. The theory of their origin which has been advanced in the paper already referred to [8] is based on the hypothesis, supported by the results of clinical experiment, that the attacks are brought about by sudden changes in blood vessels in the vestibule, changes which are conditioned by variations either in their permeability or in their caliber. If this vascular theory is accepted, at once there is a simple explanation for the variations in vestibular function in different cases, even in the same case at different times, and for the distribution of hypofunction in one group and of hyperfunction in the other.

THE ALLERGIC GROUP

In this group the vessels of the stria vascularis, from which by a process of dialysis the endolymph is presumed to be produced, dilate, the

permeability of their walls is increased and an excess of endolymph is poured out. This excess of endolymph will damp down the movement of the hair cells in the semicircular canals on the affected side until, by its increasing pressure, function is so diminished that compensation becomes impossible and an attack of vertigo results. When the attack is over, the capillary dilatation in the stria has subsided, and the pressure of endolymph will gradually subside and return to normal. Hence, in the interval period when tests are made, either some diminution of function will still remain or the status quo ante will have been reestablished and equality between the two sides will reign. The more frequent the attacks or the longer the condition has been established, the more permanent the diminution of function. Moreover, the same damping-down process which affects the vestibule will affect the cochlea also, and the whole of the cochlea, so producing the characteristic audiogram with more or less equal diminution of hearing for all tones. Additional evidence for this explanation is provided by the alteration in vestibular function which takes place as the result of treatment. Thus in example 1, in which the disease was in an early stage and in which vestibular function in the affected ear was at outset 35/60, it changed during treatment to 30/105 (1 : 3½) and at the end it was 25/120 (almost 1 : 5), a slight hyperfunction, the function of the other ear remaining all the time normal at 1 : 4.

THE VASOCONSTRICTOR GROUP

The modus operandi in this group is, on the same hypothesis, just the reverse. Here a vasoconstrictor mechanism is in operation. Whereas the cochlea is an avascular organ (Kolmer [4]) like the lens of the eye and depends for its nutrition on the endolymph, the maculae and the cristae rest on a rich capillary bed. If, by vascular spasm, the blood supply is diminished, and so function, to any considerable extent on one side as compared with the other, an attack of vertigo will result, since labyrinthine vertigo is only the expression of an imbalance between the two sides.

But why, then, the hypersensitivity of the affected labyrinth which is found in the intervals between attacks? If it is agreed that there is, or may be, a more or less permanent diminution of the blood supply to that labyrinth, whether as a result of degenerative arterial disease or of vasospasm from an overactive vasoconstrictor pathway, the explanation is not too difficult, contradictory though it may seem at first sight that even the elderly patients in this group should have an increase of function. A

4. Kolmer, W., in von Möllendorff, W.: Handbuch der mikroskopischen Anatomie des Menschen, Berlin, Julius Springer, 1927, vol. 3, pt. 1, p. 348.

sense organ to which the blood supply is temporarily diminished commonly evinces a paresthesia, a heightened awareness associated with a perverted response to stimulus. This is evident, for instance, in the limb which has "gone to sleep," in which there is not only spontaneous abnormal sensation, the characteristic "pins and needles," but at the same time a heightened response to normal stimuli, which makes it painful to stand or to grasp. This altered reaction to a normal stimulus may persist in diminishing degree for long periods even after a very minor and temporary interference with the vascular supply. It becomes easy to understand, then, how permanent might be the effect of an almost continuous interference. The vestibule may be expected to react to diminished blood supply in the same way as any other sense organ, by a paresthesia—hence, I suggest, the hyperfunction between attacks. Furthermore, the degree of the increased response to stimulus will vary with the degree of vascular spasm, and thus there is an explanation for some at least of the variation in findings. But if the diminution in blood supply persists over long periods a gradual degeneration may be expected to take place and ultimately, therefore, hyperfunction to give way to hypofunction. The 2 patients in this group who exhibited such hypofunction had suffered attacks over ten and twenty-one years.

The change in cochlear function is also explicable on this basis. If the blood supply to the vestibule is diminished, so is that to the cochlea, or more probably to the cochlear nerve, since the cochlea itself is an avascular structure. The audiogram therefore would be expected to be that characteristic of a perceptive deafness—as it is.

CONCLUSIONS

The apparently erratic results of vestibular function tests in patients with Ménière's syndrome have been shown to be explicable and orderly in the light of the realization that there are two groups of patients with Ménière's disease, owing their attacks to different causes.

The findings and the mechanism of their production have been discussed.

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INVOLVEMENT OF THE JAW JOINT IN ACUTE SUPPURATIVE OTITIS MEDIA

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Considering the close proximity of the mandibular fossa to the middle ear, it is remarkable that extension of infection from the middle ear to the jaw joint does not occur more often. It is not mentioned in any of the standard texts on otology, and the literature contains only one reference to it. This report is being made because suppuration in the mandibular fossa does occur secondary to otitis media, it must be differentiated from zygomatic mastoiditis and it requires special treatment.

Three patients with involvement of the jaw joint secondary to acute suppurative otitis media came under my care during a two year period.

REPORT OF CASES

CASE 1.—W. F., a boy of 7, was first seen Feb. 11, 1935, four days after the onset of earache on the right side. The right drum membrane was red, thickened and bulging; the temperature was 103 F., and the child had been vomiting since the day before. Paracentesis released pus under pressure, but the following day the temperature was 104 F.; so the child was sent to the hospital for observation. The next day, the sixth after the onset of the otitis media, a swelling was first noted in front of the right ear, with a point of tenderness over the mandibular joint. A roentgenogram of the mastoid showed a moderately pneumatized mastoid process without zygomatic cells. The mastoid cells were clouded, but there was no decalcification or coalescence. Two days later the swelling over the jaw joint had increased, with pain on chewing and depression of the right side of the mandible, so that the bite on the right side failed to close. A second roentgenogram revealed an increased space between the articular head of the mandible and the mandibular fossa (fig. 1). The swelling in front of the ear gradually increased until, one week after admission, it reached from the auricle to within an inch (2.5 cm.) of the outer canthus of the eye and from an inch above to an inch below the zygomatic process. There was no fluctuation, but the point of greatest tenderness was localized to a point immediately over the jaw joint. The temperature, meanwhile, had subsided only slightly and continued to reach 102 F. each day.

Because of the absence of fluctuation, the absence of displacement of the auricle downward and the absence of zygomatic cells, the presence of zygomatic mastoiditis with cortical perforation and subperiosteal abscess seemed unlikely, whereas the displacement and impaired function of the jaw indicated suppuration in the mandibular fossa. A needle was inserted along the inferior surface of the zygomatic process into the mandibular fossa, and pus was aspirated, confirming the diagnosis.

Drainage was established through a horizontal ¼ inch (0.64 cm.) cutaneous incision, through which a small sharp-pointed hemostat was inserted down to the lower edge of the zygomatic process and along its inferior surface into the mandibular fossa, constantly hugging the bone (fig. 2). The bone of the man-

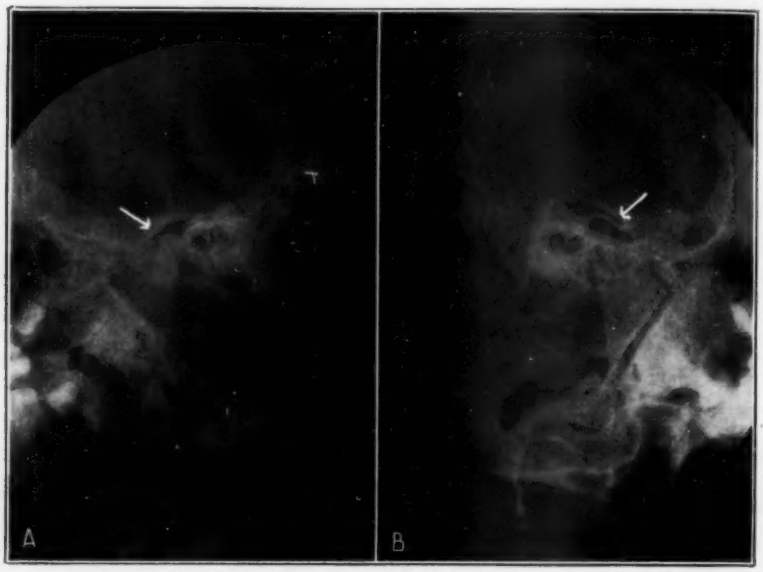

Fig. 1.—Roentgenograms of the mastoid cells in case 1 (*A*, right ear; *B*, left ear), taken one week after the onset of otitis media and two days after the onset of suppuration in the right jaw joint. Note the increased space between the articular head of the mandible and the mandibular fossa on the involved (left) side.

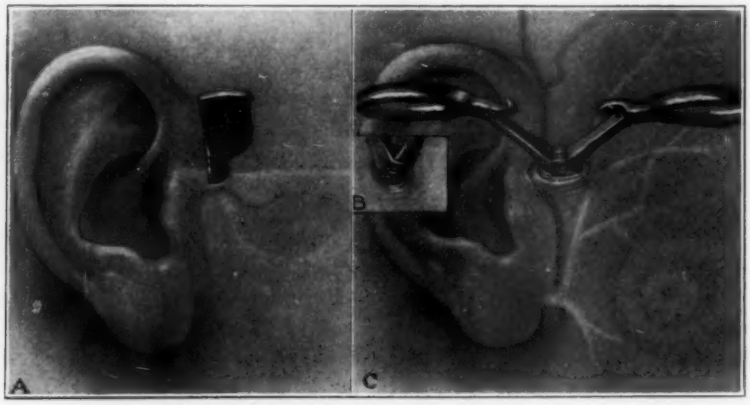

Fig. 2.—*A*, cutaneous incision used in case 1. *B*, sharp-pointed hemostat inserted down to the zygomatic process and along its inferior surface into the mandibular fossa. *C*, hemostat opened to allow the escape of pus from the abscess. Note how the superficial temporal artery is pushed aside and left uninjured.

dibular fossa was definitely roughened, and the abscess proved to be between the bone and the joint capsule. A small rubber drain was inserted into the cavity of the abscess, about 4 cc. of pus being evacuated altogether. Four days later all of the symptoms had cleared, and one week after operation the drain was removed and the child was discharged with a normally functioning jaw joint.

CASE 2.—N. F., a girl of 7, was seen March 10, 1937, three weeks after the onset of bilateral acute suppurative otitis media complicating scarlet fever. There had been constant pain in the right ear since the onset. For four days the pain experienced on opening the mouth had prevented the child from eating or drinking. Examination revealed a profuse mucopurulent discharge from both ears, with bilateral tenderness over the mastoids, more marked on the right. Over the right zygomatic root there was a large, fluctuant, tender swelling, with displacement of the auricle downward and outward. There was marked limitation of motion of the jaw joint, the bite being held open about ¼ inch (0.64 cm.), with inability to close the bite or to widen it more than a fraction of an inch farther. The lower teeth were displaced toward the left side.

The child was admitted to the Municipal Contagious Disease Hospital the same day, and a bilateral mastoidectomy was carried out. On the right side a large subperiosteal abscess was found, extending from just above the external canal to a point 2 inches (5 cm.) anterior, over the zygomatic process. No perforation of the cortex was found. The mastoid cells on both sides were markedly decalcified and coalescent and were filled with granulations and pus. It seemed probable that the subperiosteal abscess communicated with the mandibular fossa, so no further measure was taken to drain the mandibular fossa.

After the operation the limitation of motion of the jaw joint rapidly and progressively diminished until normal function was resumed nine days later. Some swelling and tenderness over the right mandibular joint were still present three weeks after operation but then disappeared.

CASE 3.—R. O., a boy of 6, was admitted to the Municipal Contagious Disease Hospital on Oct. 22, 1937, on the fourth day of an attack of scarlet fever. On November 6 he complained of pain in the right ear, and his temperature rose to 103.6 F. On November 8 the right ear was discharging. On November 14 he first complained of pain in the right eye, and for the next ten days the ear continued to discharge and the child continued to complain intermittently of pain in the right eye and right cheek. On November 30, two and a half weeks after the onset of the otitis media, a swelling was first noted over the right side of the face; the child was crying constantly with pain in this region, and his temperature was 103.8 F.

I first saw the child in consultation on December 1. There was a swelling over the region of the right mandibular joint, with the point of greatest tenderness immediately over the joint. There was marked limitation of motion of the jaw with displacement of the lower teeth to the left, and the right side of the jaw was lower than the left (fig. 3). Backward pressure on the chin caused pain in the right jaw joint. The temperature was 102.8 F. The auricle was not displaced, and there was no fluctuation. There was definite tenderness over the mastoid. A diagnosis was made of suppuration in the right mandibular fossa and probable coalescent mastoiditis.

Operation on the right mastoid December 2 revealed a well pneumatized mastoid process with extensive zygomatic development. The mastoid cells were coalescent and filled with pus and granulations, but careful search failed to disclose a perforation from the zygomatic cells into the mandibular fossa. After completion of the mastoidectomy a second incision was made over the jaw joint, as in case 1, and by means of a small hemostat hugging the bone of the inferior surface of the zygomatic process about 2 cc. of creamy pus was evacuated from an abscess lying between the bone and the joint capsule. A small rubber drain was inserted. The symptoms rapidly receded, and three days later the temperature was normal and the normal function of the jaw joint was restored. The drain was removed on the fifth postoperative day.

Fig. 3.—The patient in case 3, showing displacement of the right side of the jaw downward by the abscess in the mandibular fossa.

COMMENT

The only previous reference to involvement of the jaw joint secondary to an aural infection is in an article by Völger,[1] published in 1923. In 1 of his cases a furuncle of the external auditory canal with absence of the bony anterior canal wall resulted in a displacement of the lower jaw to the opposite side with inability to close the bite by 0.5 cm. As the furuncle subsided, the function of the jaw became normal. In Völger's other 2 cases the involvement of the jaw joint followed an acute suppurative otitis media owing, the author suggested, to extension from infected zygomatic cells. In 1 case the mandibular fossa was drained by extending the upper end of the postaural mastoid

1. Völger, G.: Ueber Miterkrankung des Kiefergelenkes bei entzündlichen Ohrerkrankungen, Ztschr. f. Hals-, Nasen- u. Ohrenh. **5**:43-47, 1923.

incision forward, and in the second case the symptoms subsided spontaneously.

To Völger's 2 cases may be added my 3 cases of suppuration in the mandibular fossa secondary to acute suppurative otitis media.

There are three possible ways in which infection might extend to the manibular fossa from the middle ear:

1. In otitic sepsis, in which metastatic foci may occur in any of the joints, but especially in the hip joint.

2. By bone erosion from infected zygomatic cells, as suggested by Völger and as probably occurred in case 3, or by extension of a subperiosteal zygomatic abscess down into the mandibular fossa, as probably occurred in case 2.

3. By thrombophlebitis of the anterior tympanic veins, which pass from the middle ear through the petrotympanic fissure to join the plexus of veins around the jaw joint. This probably occurred in case 1.

DIAGNOSIS

In diagnosing involvement of the jaw joint secondary to acute otitis media, one must differentiate this condition from furuncle of the external canal and from zygomatic mastoiditis with cortical perforation. There are eight diagnostic criteria of suppuration of the mandibular fossa which are of value:

1. Swelling over the jaw joint without displacement of the auricle.

2. Localization of the point of greatest tenderness over the jaw joint.

3. Displacement of the jaw downward on the affected side, so that the bite fails to close on this side, while the lower teeth are displaced toward the opposite side (fig. 3).

4. Pain on chewing and limitation of the motion of the jaw.

5. Pain in the jaw joint caused by pressure on the tip of the chin (compressing of the jaw joint).

6. Roentgen evidence of the widening of the mandibular joint space (fig. 1).

7. Fever and leukocytosis.

8. Pus obtained on diagnostic aspiration of the mandibular fossa.

TREATMENT

Study of the anatomy of the jaw joint shows that the mandibular fossa consists of a posterior roughened nonarticular portion and an anterior smooth articular portion (fig. 4). The smooth articular por-

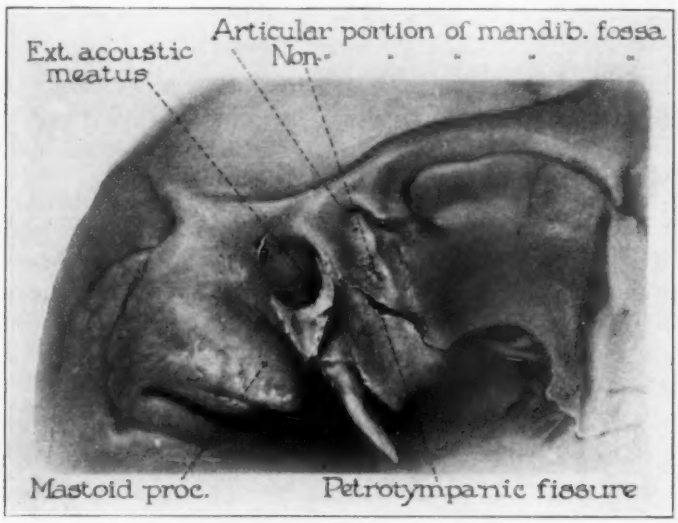

Fig. 4.—Mandibular fossa viewed from the side and below. Note the smooth anterior half of the fossa; this is the articular portion and is covered with cartilage. The posterior roughened nonarticular portion of the fossa is covered by fibrous tissue separating it from the joint capsule.

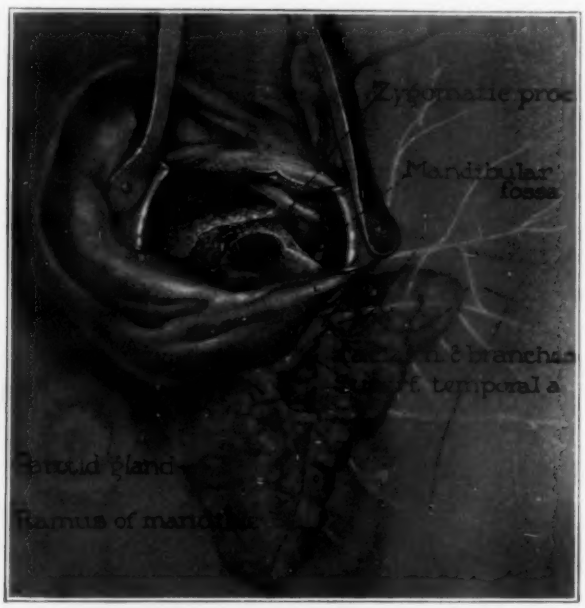

Fig. 5.—Endaural simple mastoidectomy combined with drainage of the mandibular fossa. The use of this method would avoid any scar on the face other than the usual, almost invisible, vertical scar resulting from the endaural incision. Note how this method of drainage also avoids injury to the superficial temporal artery, which normally passes directly over the jaw joint.

tion of the fossa is covered with cartilage, while the rough nonarticular portion is covered with loose fibrous tissue, which fills the space between the bone and the joint capsule. The petrotympanic fissure, through which the anterior tympanic veins and the chorda tympani nerve pass from the middle ear to the mandibular fossa, opens into the nonarticular part of the fossa. In all 3 of my cases the abscess was found to be outside the joint capsule, in the nonarticular part of the mandibular fossa. This is of importance in therapy. If conservative measures (convalescent serum and chemotherapy) do not result in a recession of the symptoms, surgical drainage should be instituted to prevent further extension through the joint capsule into the joint itself, where necrosis of the cartilage would probably be followed by ankylosis of the joint. Drainage of an abscess of the mandibular fossa must aim to avoid injury to the capsule or joint cartilages, as well as injury to the superficial temporal artery, to the parotid gland or possibly to the upper branches of the facial nerve.

The technic used in cases 1 and 3 leaves a minimum scar and avoids injury to overlying structures and to the joint capsule or cartilages (fig. 2). An alternative method of drainage that might be used with the endaural approach to the mastoid would be that of extending the endaural incision anteriorly at the junction of the cartilaginous and the bony canal for a distance of about 0.5 cm. This permits the periosteum and the soft tissues to be retracted forward until the nonarticular portion of the mandibular fossa comes into view (fig. 5). Thus a second scar is avoided in cases in which a simple mastoidectomy is required.

SUMMARY

Three cases of involvement of the jaw joint complicating acute suppurative otitis media are added to the 2 previously reported cases.

The diagnosis of suppuration of the mandibular fossa is made on the basis of clinical and roentgen findings. The treatment is surgical drainage without opening of the joint capsule.

122 South Michigan Avenue.

VASOMOTOR RHINITIS

A PROPOSED CLASSIFICATION BASED ON AN ANALYSIS OF SEVENTY-FOUR CASES

ERICH URBACH, M.D.
PHILADELPHIA

In examining 379 patients with asthma, I [1] found vasomotor rhinitis in 145 (38 per cent). The incidence was highest among those with specific allergic asthma, about 47 per cent; among those with so-called pathergic asthma it was 37 per cent. When the vasomotor rhinitis appeared at about the same time as the asthma, the conditions were brought on by the same causal agents, and when the vasomotor rhinitis appeared long before or after the asthma, the conditions were attributable to different agents.

The present contribution is an analysis of 74 cases of vasomotor rhinitis in which there was no history of asthma or any evidence of asthma at the time of examination. The patients had presented themselves for treatment only of refractory nasal conditions. They were examined in 1936 and 1937 in my allergy department in the Krankenhaus der Kaufmannschaft, in Vienna, according to a uniform plan of procedure, described elsewhere [2] in relation to asthma. In order to confirm the nasal findings, the patients were all subsequently studied in the department for nose, ear and throat diseases of the University of Vienna.

The diagnosis "vasomotor rhinitis" was made partly on the basis of subjective manifestations (paroxysmal attacks of sneezing, nasal obstruction or serous rhinorrhea, usually of short duration and not confined to any particular season) and partly on the basis of the conditions observed (turgescence and pallor of the mucous membrane). Furthermore, roentgenograms of the sinuses were taken in every case.

I feel that first of all I should make my position clear as to the nomenclature of the disease under consideration. "Vasomotor rhinitis" is a collective term, embracing a fairly wide range of clinical symptoms. Actually the symptoms named in the preceding paragraph are alike only

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From the Jewish Hospital, Service of Dr. Erich Urbach.

1. Urbach, E.: The Relation of Vasomotor Rhinitis to Bronchial Asthma, to be published.

2. Urbach, E.: Analysis of Four Hundred and Fifty-Two Asthmatic Ward Cases, Internat. Clin. **4**:89, 1940.

in that they do not develop on an anatomic basis; their causation is partly in the nose and partly in the entire organism. For these reasons the designation "vasomotor rhinitis" is now obsolete.

As is known, of course, a considerable number of terms have been suggested to designate this syndrome, e. g., "nervous rhinitis," "rhinitis hyperesthetica," "rhinitis allergica," "hydrorrhea nasalis," "perennial nasal allergy," "coryza spasmodique," "coryza allergica," "atopic coryza," paroxysmal rhinorrhea," "extrinsic rhinitis" and "intrinsic rhinitis," and "rhinopathy." Most of the proposed designations must be rejected a priori, for either they are just as vague as "vasomotor rhinitis" (for example, "nervous rhinitis," "rhinitis hyperesthetica," "coryza spasmodique," "paroxysmal rhinitis") or they are appropriate for only a small group of the cases (for example, "rhinitis allergica," "perennial nasal allergy"). At first glance, Rackemann's suggestion of dividing the cases into those of extrinsic and those of intrinsic rhinitis seems satisfactory. On more careful consideration, however, it becomes evident that intrinsic rhinitis embraces conditions caused by such heterogeneous agents as bacteria and foods and, moreover, that such agents can, under different etiologic circumstances, also produce extrinsic rhinitis.

In my opinion, the correct general designation is undoubtedly "rhinopathy," in accordance with the widely accepted usage of the suffix "pathy"—as in "hepatopathy," "nephropathy," "arthropathy" and "neuropathy." In contrast with the term "rhinitis," "rhinopathy" does not suggest an inflammatory disease. Subclassification could be undertaken along the lines of my[3] proposed division of phenomena of hypersensitivity into allergic and pathergic, as follows: When the causation is specifically allergic, one might refer to the condition as allergic rhinopathy or pollen rhinopathy, flour rhinopathy, and so on; while, on the other hand, when the causation is a nonspecific hypersensitivity, I suggest that the condition be called "pathergic rhinopathy." Elsewhere,[2] in a discussion of bronchial asthma, I have employed this sort of subclassification and shall do so here.

DEFINITION OF PATHERGY

As is well known, there are certain hypersensitivities which are definitely specific but which are not based on an antigen-antibody mechanism (e. g., light dermatoses, physical urticarias). These conditions cannot, in my opinion, be regarded as allergies, inasmuch as the term "allergy" implies an antigen-antibody reaction. Furthermore, there are hypersensitivities which are brought about by a variety of nonspecific agents (e. g., certain eczemas, certain types of asthma, cer-

3. Urbach, E.: Textbook of Allergy, to be published.

tain rhinopathies). In order to designate comprehensively all the pathologic manifestations due to an alteration in the state of reactivity, I [3] have used the general term "pathergy," [4] coined by Roessle.[5]

This gives the advantage of a logical and orderly nomenclature and clarifies concepts regarding pathologic processes of this type.

Thus the general term "pathergy" is to embrace all acquired or innate increases or decreases of reactivity of the living tissues with respect to chemical or physical agents, regardless of whether these agents are of the nature of antigens or not.

I propose the subdivision of pathergy into three main types, as follows: (1) allergic pathergy, or, in brief, allergy; (2) heteroallergic pathergy, or heteroallergy, and (3) nonallergic pathergy, or pathergy.

ORIGINAL MATERIAL

Sex and Age.—Of the 74 patients, 34 (about 46 per cent) were men and 40 (about 54 per cent) were women, with an 8 per cent predominance of women.

TABLE 1.—*Age Distribution, by Decades, of Patients with Rhinopathy*

	0-10	11-20	21-30	31-40	41-50	51-60	61-70
Males	0	1	4	18	6	0	5
Females	0	1	12	16	7	3	1

The majority of the men (53 per cent) came under my observation during their fourth decade of life, and most of the women, during their third and fourth decades (30 per cent and 40 per cent respectively). It is noteworthy that none of my patients was in his first decade of life and only 2 were in their second.

The results change somewhat when the comparison is based not on the age at the time of study but on the first recorded attack in the patient's history. Nevertheless, the third and fourth decades still show by far the highest incidence of the condition. It is to be noted that in attacks of this disease—as contrasted with those of asthma—persons in the first decade of life are almost always spared. However, this is not true in cases in which rhinopathy makes its appearance simultaneously with asthma—cases which have been deliberately excluded from consideration here. It is interesting to note that phenomena of hypersensitivity of the nose can make their first appearance in patients of advanced age. Thus, in 9 per cent of my cases, the condition first manifested itself in men in their seventies.

4. From *pathos* (Greek), disease, and *ergeia* (Greek), reactivity.

5. Roessle, R.: Die geweblichen Äusserungen der Allergie, Wien. klin. Wchnschr. **45**:609, 1932; Allergie und Pathergie, Klin. Wchnschr. **12**:574, 1933.

Race.—The fact is striking that in Vienna, where at most 20 per cent of the population was Jewish, about 50 per cent of the patients with vasomotor rhinitis (male and female about equally) were Jews. This is analogous to the high percentage of Jews (33 per cent) among patients with asthma. In my opinion, this is due not to a racial predisposition but rather to social conditions. The Jews of the present generation—and notably those in postwar Europe—have been under great psychic strain. And, as will be elaborated, the psychic status can constitute an important predisposing factor in cases of rhinopathy.

Occupation.—In regard to the incidence of rhinopathy among men according to occupation, clerks are most frequently affected, even if the percentage of the total (26 per cent) is exactly the same as that of laborers. It must be noted, however, that in Vienna there were about five times as many laborers as clerks. Most of the affected laborers were bakers, pastry cooks and millers. Farmers were scarcely ever affected. On the other hand, professional men and business men—in short, men who work with their brains—comprised a relatively high percentage (40 per cent) of the total number of patients with rhinop-

TABLE 2.—*First Appearance of Rhinopathy by Decades*

	0-5	6-10	11-20	21-30	31-40	41-50	51-60	61-70
Males	0	0	3	11	10	6	1	3
Females	0	1	2	13	18	4	2	0

athy. Among the women affected, 42 per cent were housewives and domestic servants and 25 per cent were commercial employees.

It may seem strange, at first, that professional men, clerks and housewives—persons less likely to be exposed to allergens than are men and women of the laboring class—are the more likely to suffer from rhinopathy. This will appear less paradoxic when—as I have already pointed out [2] in an analysis of the occupations of patients with asthma—the significance of psychic strain is kept in mind. Clerks are usually educated persons of a higher social level. Their reaction to low wages and their fierce struggle for existence in Europe make life harder for them than for persons of the laboring class. It must also be remembered that the housewives of postwar Germany have had to cope with the problems of regimented economy.

I do not, of course, wish to imply that these are the only factors predisposing to rhinopathy in these groups—especially in women, since the high incidence can be partially explained by the fact that the majority are housewives or domestic servants.

Duration of Rhinopathy at Time of Admission of the Patient.—More than 85 per cent of all the men and 87 per cent of all the women

had suffered from rhinopathy for more than a year before consulting me. More than 38 per cent of the men and 30 per cent of the women had suffered from the disease for longer than six years.

Family History of Allergy.—It is striking to note in how few cases there was any demonstrable family history of allergy. This may be due partially to the unusual nature of the material under consideration, as cases of rhinopathy plus asthma have been deliberately excluded. The incidence of asthma shown in the family history (19 per cent) was only a little higher than the average for the population of Vienna (8 per cent). A history of migraine was also found with relative frequency (15 per cent). However, it has not yet been determined to what extent migraine is elicited allergically and, above all, endoallergically.

Personal History of Allergy.—Strikingly few of the patients of this group with rhinopathy reported allergic symptoms—that is to say, aside from migraine; and, as was mentioned previously, the relationship between migraine and allergy is still vague. Migraine was found

TABLE 3.—*Incidence of Rhinopathy by Occupation*

	School Children	Laborers	Clerks	Housewives and Domestic Servants	Professional People	Merchants and Manufacturers	Farmers
Males.....	1	9	9	0	7	7	1
Females.....	1	6	10	17	2	4	0

in 21 per cent of the patients with rhinopathy, among whom there was a clear preponderance of females (27 per cent women, 15 per cent men). I should like to stress again that I excluded from consideration the 145 cases of rhinopathy in which the symptoms appeared along with symptoms of asthma.

Predisposing and Releasing Factors of Rhinopathy.—The question as to the factors causing rhinopathy must be considered with regard to: (1) the predisposing conditions and (2) the eliciting or releasing factors. It is evident that one must rely almost exclusively on the patient for information concerning the predisposing factors, while the releasing factors are more or less open to discovery by means of experimental testing.

In considering asthma and rhinopathy, I [2] have undertaken to divide the causes into predisposing and releasing factors, for the following reason: In cases of disease due to hypersensitivity of long standing, it frequently happens that the attack is brought on by any one of a wide variety of nonspecific releasing factors, such as wind, cold, dust, excitement or exertion. Combating of one or more of these releasing

agents therefore rarely brings any decisive results. But when it is possible to manage effectively the underlying, predisposing cause—as, for example, a tendency to colds, chronic sinusitis, extreme nervousness, or hyperthyroidism—then progress has been made toward combating the disease of hypersensitivity, if the releasing agent is taken into consideration at the same time.

As will be seen in table 5, in 44.6 per cent of all instances, the rhinopathy occurred in conjunction with a general infection (e. g., grip or measles) or with a local infection (e. g., a head cold). In 20 per cent of the cases—the second largest group in this connection—the rhinopathy was brought on by local irritation of the nasal mucosa, either by acid vapors or dust or by local operations (six times). Among the remaining cases, there are several interesting instances in which the condition appears to have been brought on by nervous factors (excitement, psychic strain) and several in which it was based on endocrine disturbance (hyperthyroidism, menopause) or an intestinal disease. I shall discuss these factors further.

TABLE 4.—*Duration of Rhinopathy at the Time of the Patient's Admission*

	Up to 3 mo.	3 to 12 Mo.	1 to 5 Yr.	6 to 10 Yr.	11 to 20 Yr.	21 to 30 Yr.	31 to 40 Yr.
Males	2	3	16	9	3	1	0
Females	1	4	23	4	5	2	1

Now as to the releasing factors: In 19 cases (in about 25 per cent of all the cases) it was possible to demonstrate specific allergic hypersensitivities. These were distributed as follows: hypersensitivity to flour, 7; to feathers in bedding, 3; to coffee, 2; to animal emanations, 2; to molds, 1; to house dust, 1; to straw dust, 1; to drugstore dust, 1, and to ingested egg, 1. In the majority of these cases the patients' contacts with the offending substances were occupational. In 28 per cent of the cases, attacks were induced by local irritation of the nasal mucosa, e. g., from cold air, sudden change in temperature, wind, and so on. In about 12 per cent of the cases, the rhinopathy always followed irritation by street dust, chemicals or irritating gases. In another group of about the same size, the condition was brought on only by an acute cold. In a third group, the rhinopathy followed psychic disturbances. In some few cases, attacks of sneezing were elicited by gastrointestinal upsets, menstruation, looking into sunlight or by physical exertion.

Tests.—Various methods were employed to identify the releasing factors. Intracutaneous tests were accepted as specifically positive only if there was a nasal reaction. Such a reaction occurred in only 5 cases

in which tests were made with molds, house dust and bacterial vaccines. In 14 of the 15 cases of specifically allergic rhinopathy nasal tests were positive, and in the 1 case of nutritional rhinopathy a nasal reaction was obtained on the administration of egg. In many other cases, it was possible to obtain effects similar to those of the releasing factors: The symptoms of rhinopathy were produced, for example, by exposing the patients to the irritation of chemicals, of dust or of sunlight or by bringing about excitement or bodily exertion.

Genesis of Rhinopathy.—On the basis of the data submitted here, I should like to group the 74 cases etiologically as follows: In 54 of the cases (73 per cent), the rhinopathies belonged to the pathergic groups—that is to say, they were caused by a variety of nonspecific factors. I subdivide these conditions, according to the primary noxae, into rhinopathy on the basis of infection, on that of irritation, on that of psychic influences and on that of a condition which formerly was

TABLE 5.—*Factors Predisposing to Rhinopathy*

Predisposing Factor	Patients
Acute cold, grip	33
Irritation by acid vapors, dust, etc.	9
Nasal operation (polyps, septum)	6
Chronic paranasal infection	5
Excitement, psychic disturbance	4
Hyperthyroidism	2
Physical exertion	2
Intestinal disease	2
Menopause	1
Unknown	10

specifically allergic. Rhinopathy on the latter basis is a condition which was originally specific but which in the course of time became nonspecific. Of the remaining 20 cases in about two thirds the rhinopathies were specifically allergic; in one sixth, they were due to endocrine disturbances, and in another one sixth, to intestinal disturbances. It has not yet been ascertained to what extent the rhinopathies based on endocrine or intestinal disturbances were of endogenous allergic or toxic origin.

Treatment.—Just as in the case of asthma, there is no standard method of treatment for rhinopathy. Whenever possible, each patient should be treated etiologically. Unfortunately, this approach is hardly possible with the majority of the patients with pathergic rhinopathy. Operations—even operations as drastic as those of Caldwell and Denker—are usually without lasting effect; occasionally they are even harmful. Thus, of our 74 patients, 21 had undergone the greatest variety of nasal operations, practically without any benefit.

In my opinion, the best approach in the treatment of pathergic rhinopathy is, on the one hand, to lower the usually nonspecific hyper-

sensitivity of the nasal mucosa and, on the other, simultaneously to increase the patient's resistance. A combination of methods is necessary. Repeated small doses of phenobarbital, 1/8 to 1/4 grain (0.0075 to 0.015 Gm.) daily, lower the general irritability of the patient. This treatment is to be continued for weeks. In order to accustom the nasal mucosa to local irritation, appropriate breathing exercises (humming while standing, later while running) of three to ten minutes' duration should be practiced three times daily. I have seen good results following the administration of small doses of roentgen ray (150 r with filters of 0.3 mm. copper + 1 mm. aluminum, three times at intervals of one month). When the rhinopathy is of an infectious nature, autovaccine should be prepared whenever possible and should be administered weekly in ascending doses if there is a positive cutaneous reaction. Almost all patients with pathergic rhinopathy react strikingly to old tuberculin. I therefore also give a weekly *intracutaneous* injection of a small dose, beginning with 0.1 cc. of a dilution of 1 : 1,000,000

TABLE 6.—*Releasing Factors of Rhinopathy*

Releasing Factor	Patients
Cold air, sudden change in temperature, wind	21
Acute cold	9
Specific allergen (flour, animal emanation, egg, etc.)	19
Irritation by chemicals, dust	9
Excitement, psychic disturbance	8
Physical exertion	1
Sunlight	2
Constipation, gastrointestinal upset	3
Menstruation	2

of old tuberculin in cases in which this amount elicits a clearcut reaction. Furthermore, general strengthening and tonic measures should be prescribed, such as baths with subsequent cold showers and massage.

In the treatment of specific allergic rhinopathy, the measures to be considered are either those of elimination or those of desensitization.

Rhinopathy based on endocrine disturbances must be treated by means of endocrine substitution therapy. Hyperthyroidism must be treated appropriately.

In cases of intestinal rhinopathy, the constipation or other underlying disturbance must be removed.

I do not wish to claim by any means that my methods of treating rhinopathy are successful in every case. I do believe, however, that a great percentage of my patients have been definitely benefited by the aforementioned therapeutic measures.

I am of the opinion that every asthmatic patient, and others with a severe and not readily manageable allergy should be hospitalized. I also sincerely believe that this should be done with patients suffering

from severe rhinopathy. Hospitalization of the patient enables physicians to perform all necessary examinations as accurately and as quickly as possible.

Of the 74 patients in my series, 43 (or 58 per cent) were hospitalized. Twenty-five per cent remained in the hospital one week; 21 per cent, ten days; 35 per cent, two weeks; 16 per cent, three weeks, and 3 per cent, four weeks.

COMMENT

It now appears more important than ever to stress the point that in the majority of cases hypersensitivity of the nasal mucosa is not based on an antigen-antibody reaction—i. e., it may not be designated as "allergic." For the purpose of clearly expressing the nonspecific nature of the hypersensitivity in this kind of rhinopathy, I have suggested the designation "pathergic rhinopathy."

TABLE 7.—*Classification of Rhinopathy According to Genesis*

Classification	Patients	
	Number	Per Cent
Pathergic, on basis of infection	30	40.5
Pathergic, on basis of irritation	15	20.3
Pathergic, on basis of psychic disturbance	4	5.4
Pathergic, on basis of specific-allergic rhinopathy	5	6.7
Allergic	14	19.0
Endocrine	3	4.0
Intestinal	3	4.0

To be included under pathergic rhinopathy are: the paroxysmal attacks of sneezing with subsequent swelling of nasal mucosa, following exposure to sudden changes in temperature; attacks of sneezing attributable to hypensensitivity to cold or heat (Duke [6]); sneezing brought on by looking into bright sunlight (Duke [7]; Freund [8]), and attacks based on psychic disturbances.

In another group—as Adlersberg and Forschner [9] first pointed out—it is possible to establish a relationship between an intestinal disturbance (e. g., constipation) and rhinopathy. Elimination of the intestinal condition will alleviate the rhinopathy.

6. Duke, W. W.: Clinical Manifestations of Heat and Effort Sensitiveness and Cold Sensitiveness, J. Allergy **3**:257, 1932.

7. Duke, W. W.: Light Sensitiveness, Radiology **4**:279, 1925.

8. Freund, L.: Ueber das Niesen. Beitrag zur Photophysiologie, Strahlentherapie **21**:518, 1926.

9. Adlersberg, D., and Forschner, L.: Rhinopathia vasomotoria und Verdauungsstörungen, Ztschr. f. Hals-, Nasen- u. Ohrenh. **20**:250, 1928; Ein Beitrag zur Klinik der Rhinitis vasomotoria, Monatschr. f. Ohrenh. **4**:397, 1932.

These same authors have described, aside from enterogenic rhinopathy (as they have named the aforementioned condition), an incretoric rhinopathy. In this condition, the nasal manifestations are attributable to internal secretory disturbances. Appropriate substitutional endocrine therapy will benefit rhinopathy of this type. The attacks usually represent functional disturbances of the ovaries or of the thyroid gland.

According to Goetz [10] and Hedderich [11], oxyuriasis is a cause of rhinopathy in some cases. Furthermore, one must always bear in mind the possibility of mechanical causation by sharp-edged substances, such as the blossoms of the royal palm and the plane tree (Benjamins [12]), or barley dust.

Aside from the cases in which the state of hypersensitivity of the nose is pathergic, there are also numerous ones in which the rhinopathy is of allergic origin (hay fever will not, of course, be considered here).

In identifying the responsible agent in cases of specific allergic rhinopathy, it is well to recognize that the skin tests, whether negative or positive, have only limited value. In my opinion, a cutaneous reaction to a test substance may be considered specific only when it is accompanied by a reaction at the focus in the form of an acute rhinorrhea—a reaction which I have frequently observed. However, I attach far more importance to the results of nasal testing. Even more valuable are the tests of exposure and elimination.

First of all, one must look for allergens of exogenous origin. Secondly, food allergens are to be considered. In the third place, drugs, either orally or parenterally administered, are occasionally the cause of an allergic rhinopathy.

In the fourth place, in some cases rhinopathy may be assumed to have a bacterial allergic basis. It is true that rhinopathy can result when a chronic state of infection increases the responsiveness of the neurovasomotor and exudative mechanisms of the nasal mucosa to multiple, nonspecific irritation or when an infection predisposes to hypersensitivity to a specific allergen. Nevertheless, the investigations of Lakos [13] and my own study point to the fact that in many cases a genuine bacterial allergy may be assumed to be the cause of rhinopathy. I am inclined to make the diagnosis of allergic rhinopathy in cases in which the foci of infection are in the sinuses, the middle ear

10. Goetz, H.: Beiträge zur Kenntnis der Überempfindlichkeit, Med. Klin. **23**:1853, 1927.

11. Hedderich, L.: Klinsche Beobachtungen an 150 Fällen von Rhinitis vasomotoria, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:429, 1933.

12 Benjamins, C. E.: Hayfever (Pollenosis) in India, Geneesk. tijdschr. v. Nederl.-Indië **72**:1016, 1932.

13. Lakos, T.: Vasomotor Rhinitis as a Bacterio-Allergic Disease, Acta otolaryng. **17**:400, 1932.

or the tonsils and in those in which the removal of these foci by means of operations, the administration of sulfanilamide or other therapy brings about a cessation of the nasal symptoms. Further confirmation for this diagnosis is found in those cases in which autovaccine, taken from these foci of infection, elicits not only a strongly positive cutaneous reaction but also a nasal reaction in the form of an attack of sneezing.

SUMMARY

Vasomotor rhinitis is based on hypersensitivity of the nasal mucosa. In the minority of cases this hypersensitivity is allergic—that is to say, it is based on antigen-antibody reaction. I suggest the term "allergic rhinopathy" for this condition. Allergens to be considered are: exogenous substances—those exerting an influence on the nasal mucosa and foods or drugs taken by mouth—and endogenous substances, such as products of the endocrine glands, of metabolism or of bacteria of the intestines.

I propose the term "pathergic rhinopathy" to designate rhinopathy based on nonspecific hypersensitiveness. This condition can be the result of an underlying general or nasal infection, of a local irritation, of psychic disturbances and also of a nonspecific broadening of the bases of a hypersensitivity which was formerly specifically allergic.

An analysis of 74 cases, which were studied from the point of view of etiology, pathogenesis and therapy, is presented and forms the foundation of the proposed classification of the "rhinopathies."

SURGICAL AND NONSURGICAL CARE OF THE CHRONICALLY DISCHARGING MIDDLE EAR

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A cure for chronic discharge from the ears has been sought throughout the ages. The ancients Hippocrates [1] (460 B. C.) and Celsus [2] (30 B. C.) frequently mentioned aural suppuration and warned of the dangers which arise therefrom. In spite of these early observations, it was not until the latter part of the nineteenth century that any scientific knowledge concerning the ears and their treatment was recorded.

A possible reason for the long neglect of this subject by medical men is given by Harvey Graham in his "Story of Surgery," in which he tells of an Englishman, John of Gaddesden,[3] born in 1280, who vaguely mentioned certain diseases of the ear and recommended that all morbid material in the ears be sucked out through a tube. Graham then added, "small wonder that surgeons of succeeding centuries, tough stomached though they were, left their patients' ears to the quacks."

Eustachius [4] in the sixteenth century and Du Varney [5] and Valsalva [6] in the early eighteenth century added much to the knowledge of the anatomy and physiology of the ear but little to the treatment. To Toynbee in England about 1850 and to Adam Politzer in Germany twenty years later must be given the credit for rescuing otology from the quacks and establishing it as a legitimate branch of surgery. Jean Louis Petit [7] (1736) was the first deliberately to open the mastoid bone for the cure of suppuration, but it was not until one hundred and fifty years later that the operation was accepted.

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1. Hippocrates, cited by Ballance, C. A.: Surgery of Temporal Bone, New York, The Macmillan Company, 1919, vol. 1.

2. Celsus, cited by Ballance, C. A.: Surgery of Temporal Bone, New York, The Macmillan Company, 1919, vol. 1.

3. John of Gaddesden, cited by Graham, H. (pseud. of I. H. Flack): The Story of Surgery, New York, Doubleday, Doran & Company, 1939, pp. 377-378.

4. Eustachius, cited by Curtis, J. H.: An Essay on the Deaf and Dumb, ed. 2, London, Longman, Rees & Co., 1834.

5. Du Varney, cited by Curtis, J. H.: An Essay on the Deaf and Dumb, ed. 2, London, Longman, Rees & Co., 1834.

6. Valsalva, cited by Curtis, J. H.: An Essay on the Deaf and Dumb, ed. 2, London, Longman, Rees & Co., 1834.

7. Petit, J. L.: Traité des maladies chirurgicales, Paris, T. F. Didot jeune, 1774, vol. 1, p. 189.

Within thirty years (1861 to 1891), a group of German surgeons, notably von Tröltsch,[8] Jacoby,[9] Schwartze and Eysell,[10] Küster,[11] Zaufal[12] and Stacke,[13] worked out the indications and perfected the technics of the various types of mastoid operation. So accurate and so complete was this work that their recommendations stand as classic today. Prior to their time, treatment of chronic otorrhea was entirely medical and was limited to the application of counterirritants in the form of blisters, leeches, setons and liniments, supplemented by the doubtful practice of instilling strong solutions of silver nitrate into the aural canals.

Since the beginning of the present century, many methods of treatment have evolved. These vary from measures for keeping the canal dry and clean to the classic radical mastoid operation.

During the past two decades great progress has been made, as shown by the marked drop in the percentage of cases of chronic purulent otitis in relation to the population of the United States. Some of the more important factors in the production of this happy state of affairs are:

1. Education of the public to the dangers of neglect and to the necessity for early and adequate treatment of all acute aural infections.

2. Realization by the members of the medical profession that acute involvement of the mastoid requires operation not only to save life but also to save hearing and to prevent chronic involvement of the mastoid.

3. Removal of infected tonsils and hypertrophic adenoids and treatment of acute infections of the upper respiratory system during childhood.

4. Better national living conditions; more accessible clinics and trained specialists.

5. Better supervision of the milk supply with consequent diminution in the incidence of aural tuberculosis (bovine type), so frequently seen in young children in the past.

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8. von Tröltsch: Ein Fall von Anbohrung des Warzenfortsatzes bei Otitis interna mit Bemerkungen über diese Operation, Virchows Arch. f. path. Anat. **21**:295, 1861.

9. Jacoby, L.: Zur Perforation des Warzenfortsatzes, Arch. f. Ohrenh. **4**: 212, 1869.

10. Schwartze, H., and Eysell: Ueber die künstliche Eröffnung des Warzenfortsatzes, Arch. f. Ohrenh. **1**:157, 1873.

11. Küster, E.: Ueber die Grundsätze der Behandlung von Eiterung in Starrwandige Höhlen, mit besonderer Berücksichtigung des Empyems der Pleura, Deutsche med. Wchnschr. **15**:185, 213, 233 and 254, 1889.

12. Zaufal, E.: Zur Geschichte und Technik der operative Freilegung der Mittelohrräume, Arch. f. Ohrenh. **37**:33, 1894.

13. Stacke, L.: Betrachtungen über den gegenwärtigen der Therapie chronischer Mittelohreiterungen, Berl. klin. Wchnschr. **26**:350, 1889.

6. Use of sulfanilamide and sulfapyridine (2-[paraaminobenzene-sulfonamido]-pyridine) in the treatment of acute otitis. (Careful observation is necessary after the use of these drugs, however, since acute infections so treated frequently drift into chronic conditions giving only the mildest of symptoms.)

Encouraging though the improvement has been, it is a regrettable fact that there are still many acute aural infections which are allowed to become chronic through neglect and which remain disturbing factors because of improper subsequent treatment.

SYMPTOMS

In chronic purulent otitis media, the discharge may continue for weeks, months or years, may be continuous or intermittent, profuse or scant, and may be with or without odor. It usually occurs without pain or fever. The only complaints may be otorrhea and impaired hearing. Perforation of the drum membrane is always present; the size of the perforation bears no relation to the degree of deafness or to the extent of the pathologic process. The location of the perforation, however, gives a definite indication of the underlying morbid condition. Central perforations usually mean involvement of the mucous membrane only and are not dangerous; marginal or attic perforations usually mean involvement of the bone and are dangerous.

CONSERVATIVE, OR NONSURGICAL, TREATMENT

While no precise line of demarcation between acute and chronic suppuration exists, a basis for proper treatment of purulent otitis media can be determined by considering the pathologic conditions which lead to chronicity. These may be classified as follows:

1. Poor hygiene and poor resistance.
2. Infections and obstructions in and about the eustachian tube.
3. Allergy.
4. Infection of the mucous membrane of the middle ear without involvement of bone.
5. Chronic mastoiditis.

Poor Hygiene and Poor Resistance.—Poor living conditions, improper food and lack of sunshine mean lack of resistance. A state of low resistance may also result from some intercurrent disease, such as tuberculosis, anemia, diabetes or nephritis.

In many cases chronic otorrhea readily responds to improved living conditions combined with proper vitamin therapy.

It usually improves in proportion to the improvement of the general physical condition of the patient.

Infections and Obstructions in and About the Eustachian Tube.—The eustachian tube is the route by which many acute infections reach the middle ear and is not infrequently the pathway by which suppurative processes in the middle ear are maintained from foci adjacent to the nasopharynx. Complete eradication of all growths, infections and obstructions in the nasopharynx, nose, nasal accessory sinuses, tonsils and teeth is the first step in the treatment of otorrhea of this type. No case in which there is otorrhea should be classified as one of chronic purulent otitis media while such infections and obstructions are present.

Allergy.—Little has been written about and little attention has been given to the relationship between the allergic state and chronic middle ear discharge. The reactivation of old suppurations of the middle ear by acute allergic manifestations in the nose and nasopharynx is a common occurrence. Allergy is many times the cause of discharge from the middle ear and when it is perennial may be the cause of continuous suppuration.

When the secretion is excessive and mucoid allergy should always be suspected. A cytologic examination of the nasal secretion for eosinophils according to the Hansel [14] method will frequently confirm the diagnosis.

Control of the allergy gives universally good results not only in diminishing the discharge but also in improving the hearing.

Infection of the Mucous Membrane of the Middle Ear Without Involvement of Bone.—In this simple type of purulent otitis media the infection is confined to the mucous membrane of the middle ear and eustachian tube and occasionally of the epitympanic region. The establishment of adequate drainage is the first prerequisite to successful treatment. This means removal of all obstructing polyps and granulations and the enlargement of the perforation if necessary, to insure proper drainage and allow the introduction of medication to the middle ear. Small areas of granulation can be destroyed by touching them with toughened silver nitrate (silver nitrate sticks) or trichloroacetic acid and larger areas by means of a curet or, better, by electric coagulation. Large polyps can be removed with the aural snare, care being taken to avoid jerking them away, since they are occasionally attached to the stapes or other small bones of the ear.

In the treatment of middle ear infections of these first four classes scrupulous cleanliness is most important. Indiscriminate douching of the ear is condemned by most otologists. Excessive secretions are best removed by suction. If swabs are to be used, they should be sterile. The common practice of making swabs by winding sterile cotton on

14. Hansel, F. K.: Personal communication to the author.

wire applicators with unsterile fingers cannot be too strongly condemned. The middle ear should be treated as an open wound, most susceptible to outside infection.

The use of dehydrating drops and irritating solutions in the treatment of these simple types of otorrhea is counterindicated. The middle ear and the eustachian tube are lined with mucous membrane, which is normally moist, and any treatment such as frequent irrigations with irritating antiseptics, the insufflation of caustic dusting powders or the introduction of dehydrating solutions like 95 per cent alcohol is not rational and physiologic. One would hesitate to apply the same medication to the mucous membrane of the nose in an effort to cure chronic rhinitis. Repeated applications of such caustic substances, while possibly having a drying effect on the ear, will destroy any remaining normal mucosa of the middle ear and cause scar tissue to take its place, with resultant irreparable damage to hearing.

Instillation of 0.25 per cent isotonic solution of neo-synephrin hydrochloride buffered to p_H 6.7 into the middle ear with the head held in a position favorable for the entrance of the solution into the eustachian tube will do much to open the tube, stimulate the cilia and reestablish drainage. The middle ear should be thoroughly dried with sterile swabs and warm compressed air after this treatment.

Oral administration of capsules containing ephedrine, phenobarbital and atropine is of great value in maintaining the patency of the tube and in lessening the secretion.

The patient should be cautioned against allowing water to enter the middle ear through the external canal, and careful instructions should be given as to the proper method of blowing the nose.

By following these simple principles of treatment one generally obtains satisfactory results. In these uncomplicated types of middle ear infection a mastoid operation is seldom indicated, since involvement of bone and complications are rare.

Chronic Mastoiditis.—In the dangerous type of chronic purulent otitis media, the perforation is marginal or into the attic; either of these conditions usually means involvement of bone and is conducive to the formation of cholesteatoma. Since cholesteatoma is by far the commonest cause of labyrinthian and intracranial complications, the patient's life is in danger as long as the condition is present. Conservative treatment for this type of infection is permissible only in the absence of all symptoms of extension of the infection beyond the limits of the middle ear, attic or antrum.

The treatment of the dangerous type of chronic mastoiditis will be discussed under the following headings:

1. Establishment of adequate drainage.
2. Removal of morbid material.

3. Application of antiseptics.

4. Roentgen therapy.

1. Establishment of adequate drainage: In the treatment of this type of infection the establishment and maintenance of good drainage from the middle ear is even more important than in the treatment of infections of the simple type. All obstructing granulations, polyps and masses of cholesteatoma must be removed and if necessary the perforation enlarged. Prevention of reinfection of the middle ear by way of the eustachian tube is most important.

2. Removal of morbid material: Many technics have been outlined by various otologists. The following routine has given good results: The middle ear is syringed with 95 per cent alcohol through an attic cannula, and ether is instilled for ten minutes; a saturated solution of urea is then instilled for twenty minutes. After the middle ear has been syringed with warm Ringer's solution, the ear is thoroughly dried and iodine–boric acid powder is insufflated.

Mertins [15] was the first to advocate the use of urea as a solvent for dead protein material in chronically discharging ears. In a recent article Seltzer [16] reported that none of his patients showed benefit after the use of urea, and he concluded that in the treatment of chronic aural infections this substance is valueless. My experience, however, has been that urea preceded by ether, which is relatively nonirritating to the normal mucous membrane and which is an excellent solvent for lipoids, is efficacious in dissolving the residue of dead protein.

Several other digestants have been favorably reported. Harris [17] reported good results following the use of a freshly prepared solution of the extract of Carica papaya, and Levine [18] advised the use of a preparation of the glands of the stomach (enzymol) and dilute hydrochloric acid (10 per cent).

Some years ago I saw a patient at the ear clinic of the Stanford University School of Medicine. His complaint was that his right ear had been discharging since childhood and that he had had severe pain in the ear for the preceding two weeks. Examination revealed the entire external canal to be filled with live maggots. After the maggots had been removed from the external canal, a perforation into the attic was found, from which maggots continued to drop for three days. At the end of that time the ear was dry, clean and odorless and remained so

15. Mertins, P. S., Jr.: Use of Urea in Treatment of Chronic Otitis Media, Arch. Otolaryng. **26**:509 (Nov.) 1937.

16. Seltzer, A. P.: Report of Use of Urea in Suppurative Conditions About the Ear, Ann. Otol., Rhin. & Laryng. **49**:119, 1940.

17. Harris, R.: A Treatment of Chronic Purulent Otitis Media, Arch. Otolaryng. **26**:522 (Nov.) 1937.

18. Levine, M.: Practical Otology, Philadelphia, Lea & Febiger, 1927, p. 268.

for ten days. I then filled the ear with ether. The patient jumped from the table and ran from the clinic. Unfortunately, I have not seen him since.

In 1929 Dr. Baer [19] read his report on viable antiseptics and reported his observation that osteomyelitis wounds healed more quickly and more normally in the presence of live maggots. He advanced the theory that maggots not only eat dead protein, including dead bone, but also form some sort of enzyme which stimulates the formation of healthy granulation tissue.

Were it not for the difficulty of obtaining live sterile maggots and the wounding of the patient's esthetic sense, antiseptic treatment with this viable agent might be most effective in cases of osteomyelitis of the mastoid bone, where space is so limited. The active principle in the digestant material excreted by live maggots is thought to be allantoin, and this substance should be tried in these cases.

Regardless of the digestants used, the middle ear must be thoroughly dried, since moisture causes swelling of a cholesteatoma and stimulates its extension.

3. Application of antiseptics: Innumerable antiseptics have been advocated for use in the treatment of chronic purulent otitis media. Hett [20] and his co-workers reported cures in 75 per cent of their cases with zinc ionization. Mossböck [21] reported good results with chlorine gas. Methylrosaniline and merthiolate jelly have been recommended. Lederman [22] reported excellent results with Sulzberger powder (boric acid with iodine). This powder has been very effective in my experience.

4. Roentgen therapy: While roentgen rays are of little value in diagnosis, because of the sclerosis so frequently present in cases of chronic purulent otitis media, considerable difference of opinion exists as to their value as a therapeutic agent. Luciman [23] expressed enthusiasm regarding their use. He stated that the effect of roentgen radiation

19. Baer, W. S.: Treatment of Chronic Osteomyelitis with the Maggot, J. Bone & Joint Surg. **13**:438, 1931.

20. Hett, G. S.; Wells, A. G., and Levick, G. M.: Ionization in Suppuration of the Middle Ear, Am. J. Phys. Therapy **6**:375, 1929; cited by Kopetzky, S. J.: Acute and Chronic Otitis Media and Sinus Thrombosis, Arch. Otolaryng. **12**:377 (Sept.) 1930.

21. Mossböck, F.: Die Behandlung chronische Mittelohreiterung mit Chlorgas, Ztschr. f. Hals-, Nasen- u. Ohrenh. **38**:158, 1935.

22. Lederman, M. D.: Twenty Years' Experience with Iodine Powder in the Conservative Treatment of Aural and Nasal Suppuration, Laryngoscope **47**:110, 1937.

23. Luciman, J. H.: Treatment of Otitis Media and Mastoiditis by the Roentgen Ray, Am. J. Roentgenol. **36**:946, 1936.

on the tissues affected by chronic otitis media is the same as it is on tuberculous glands and actinomycotic tissues, that it causes hyperemia, improves circulation and breaks down and causes absorption of chronic fibrous tissue. Roentgen radiation, he claimed, destroys lymphocytes, promotes phagocytosis and liberates antibodies, enzymes and other unknown principles which promote autolysis and liquefaction. He reported remarkable results in 11 cases of chronic otitis media in which the treatment was used.

Brown [24] and his associates reported equally remarkable results in 6 cases.

Roentgen therapy in my hands has led only to disappointment

SURGICAL TREATMENT

How long to continue medical treatment, when to operate and what operation to perform are decidedly matters of surgical judgment; no set rule holds for all cases. The progress of each infection must be the deciding factor.

In the past few years there has been a growing tendency toward conservatism in the treatment of chronic otorrhea. Much more consideration is being given to the conservation of hearing. Formerly the mere presence of a chronic otitic discharge was considered a definite indication for a radical mastoid operation. Recent experience, however, has proved that there are many qualifications to this, even though the possibility of intracranial complications is well recognized. In fact, conservatism has recently been so stressed that the otologist, in his desire to spare his patient the inconvenience of an operation, is apt to continue medical treatment to the point of danger, forgetting that there are simple surgical procedures which are effective.

Once surgical intervention has been decided on, the extent of the necrosis encountered will determine how radical an operation is necessary. The three generally accepted types of operation are:

1. Complete simple mastoidectomy.
2. One of the modified radical operations.
3. The classic radical operation.

The operation of choice is the one which will remove danger to life and still leave as much of the conducting apparatus as possible, provided that the otologist always bears in mind that symptoms of an intracranial complication require immediate radical mastoidectomy and that in such a case the preservation of hearing is of secondary importance.

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24. Brown, J. P.: Roentgen Therapy in Acute and Chronic Otitis Media, Am. J. Roentgenol. **42**:285, 1939.

Complete Simple Mastoidectomy.—When there is little damage to the drum and the ossicles are intact, the operation indicated is a complete simple mastoidectomy. A satisfactory technic is as follows:

The usual incision is made through the soft parts to the cortex, the cortex is opened by means of a gouge and a mallet and the mastoid antrum and the additis are located. The mastoid cells are thoroughly removed, special attention being given to the zygomatic cells, the tip cell and those along the facial ridge. The mastoid cavity is coned out, with the antrum as the apex of the cone; all overhangs are removed. The additus is carefully enlarged by means of a curet. The wound is flushed with hot saline solution and hydrogen peroxide. Soft rubber drains are placed in the cavity, one directly into the antrum. The wound is partially closed with an interrupted suture of silkworm gut, which approximates not only the edges of the skin and the soft tissues but also the edges of the periosteum. Clysis is given during the operation. Covering the skin about the operated area with sterile petrolatum makes the first dressing practically painless. The use of rubber tubing for drains makes subsequent dressings also painless.

Modified Radical Mastoidectomy.—Various modifications of the mastoid operation have been suggested, notably those by Heath,[25] Bondy,[26] Jansen Bayer, Jenkins, Gutteridge[27] and Blackwell.[28] All of these modifications aim at removing as much of the diseased tissue surrounding the middle ear as possible without disturbing the ossicles or the membrana tympani. The operation is indicated when the involvement of the drum and ossicles is slight and the hearing remains good.

It is my opinion, however, that if a complete simple mastoidectomy does not effect a cure and the hearing is poor, radical mastoidectomy is the operation of choice, since it is the only operation which insures the patient against intracranial complications.

Radical Mastoidectomy.—If any signs or symptoms of intracranial complications, such as localized headache, vertigo, absence of response from the labyrinth, facial palsy or a positive fistula reaction, develop, the surgeon should insist on immediate radical mastoidectomy. A satisfactory technic is as follows:

A complete simple mastoidectomy is first performed. The entire posterior wall of the canal is then lowered, down to the floor of the aditus, so that no facial ridge remains; the bony bridge between the middle ear and the mastoid antrum is removed; any remnants of the incus and the malleus are removed, care being

25. Heath, C. J.: The Cure of Chronic Suppuration of the Middle Ear Without Removal of the Drum or Ossicles, or the Loss of Hearing, with Ten Cases, Lancet **2**:353, 1906.

26. Bondy, G.: Totalaufmeisselung mit Erhaltung von Trommelfell und Gerhörknochelchen, Monatschr. f. Ohrenh. **44**:15, 1910.

27. Gutteridge, E.: The Extended Schwartze Operation, M. J. Australia **1**: 769, 1938.

28. Blackwell, H. B.: A Conservative Operation for Chronic Mastoiditis, Tr. Am. Laryng., Rhin. & Otol. Soc. **41**:488, 1935.

taken not to disturb the stapes. The tip of the mastoid is lowered, allowing the soft parts partially to fill the lower portion of the mastoid cavity. All granulations are carefully curetted from the middle ear. The upper end of the eustachian tube is thoroughly curetted for about 1 cm. Bone chips are inserted tightly into the curetted area of the tube and held in place by a piece of temporal muscle and periosteum.

This technic was first employed on a patient whose eustachian tube had an opening 0.5 cm. in diameter. The result was so satisfactory that the same method was used in 4 subsequent radical mastoidectomies. The tubes in all these cases have remained closed, as proved by the fact that air cannot be forced into the middle ear through the tube.

The Y-shaped incision is used in the plastic operation. The cartilage is not removed, nor are the flaps sutured back. A soft rubber tube, from 0.75 to 1 cm. in diameter, is inserted into the external auditory canal, down to the posterior wall of the middle ear, and cut flush with the external auditory meatus; the soft parts are allowed to close about it. The mastoid cavity is loosely packed with one strip of 0.5 inch tape packing through the tube. This is removed at the end of twenty-four hours. No further packing is used. The mastoid cavity is allowed to granulate as rapidly as possible; granulations in the middle ear are kept down by electrocoagulation. The tube is removed from time to time for cleaning, and a slightly smaller one is inserted if the meatus seems too large. It is kept in place until epithelization of the canal has occurred. The posterior wound is completely closed with a subcuticular stitch after the periosteal edges have been approximated. Sulzberger powder (boric acid with iodine) is blown into the cavity at frequent intervals.

No pocketing has thus far occurred.

This procedure has the following advantages:

1. Dressing is painless, which makes the operation particularly useful in the treatment of children requiring radical mastoidectomy.
2. Convalescence is rapid.
3. Danger of the formation of postauricular fistulas is minimized.
4. The mastoid cavity is small and easily cleansed.
5. A large round meatus results, allowing wide space for inspection of the cavity.
6. Complete and permanent closure of the eustachian tube is assured.
7. A high percentage of dry ears results.

SUMMARY

Neglect of acute infections of the middle ear is pointed out as an etiologic factor in the development of chronic purulent otitis media.

Two types of infection are discussed: the dangerous and the non-dangerous.

The danger of chronicity following the use of sulfanilamide and sulfapyridine in acute infections is mentioned.

The use of irritating solutions and powders in the treatment of the nondangerous type of chronic otitis is condemned, as is the employment of unsterile swabs.

The importance of allergy as a cause of acute exacerbations is stressed.

A method of treating simple chronic infections of the middle ear is outlined.

The use of various digestants, of chemicals, of antibacterial agents and of roentgen rays is discussed.

The graduated choice of operations is recommended.

A technic for simple mastoidectomy and one for radical mastoidectomy are described.

Excellent results following the use of bone chips in permanently closing the eustachian tube are reported.

384 Post Street.

EXPERIENCES WITH ENDAURAL SURGERY

PRELIMINARY REPORT

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In his paper on the "Endaural, Antauricular Surgical Approach to the Temporal Bone," Lempert [1] concluded:

> A careful comparative study of the postoperative course, the convalescence, the period of healing and the ultimate end results obtained in the 1,780 consecutive cases in which operation was performed by me through the endaural, antauricular surgical approach . . . more than justifies my abandonment of the postauricular approach to the temporal bone.

With a view to investigating this method of approach, we employed the endaural route in operating on a small number of patients who entered the otolaryngological service of the Massachusetts Memorial Hospitals from January to July 1940, and compared the postoperative course and results with those in a similar group of cases in which operation was performed by the postaural approach. It is not our purpose in this paper to discuss the anatomic and surgical details of the method (for a discussion of these the reader is referred to the comprehensive writings of Lempert,[1] Wishart [2] and others) but rather to summarize briefly our experiences in this small series of cases.

Twenty-three patients were operated on by the endaural approach. Simple complete mastoidectomy was done on 8 of these patients, modified radical mastoidectomy (antroatticotomy) on 3, radical mastoidectomy on 5 and the endaural fenestration operation for otosclerosis on 7. In all of these operations, Dr. Lempert's technic was carefully followed.

In comparing the antauricular, endaural route with the postaural approach, certain considerations come to mind. These are:

1. The problem of adequate exposure.
2. The "operating time."

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1. Lempert, J.: Endaural, Antauricular Surgical Approach to the Temporal Bone, Arch. Otolaryng. **27**:555-587 (May) 1938.

2. Wishart, D. E. S.: The Endaural Route, Laryngoscope **49**:943-965 (Oct.) 1939.

3. The postoperative care and the duration of convalescence.
4. Complications.
5. Results.

1. The exposure of the surgical field in all the cases in which the technic advocated by Dr. Lempert was used was amazingly adequate. We were impressed with the ease of entering and exposing the antrum. While we did not have to deal with any complications of mastoiditis, such as lateral sinus thrombosis, petrositis, labyrinthitis or brain abscess, we nevertheless felt that all parts of the temporal bone were surgically accessible. It appeared that should any condition requiring surgical approach to the lateral sinus or to the middle or posterior fossa of the skull present itself it could be handled with facility through the endaural approach. We are in entire accord with Dr. Lempert's conception that for radical mastoid operations and for the fenestration operation the endaural approach "offers to the otologist a fuller and more direct view of the surgical field and more ready accessibility to it."

2. The "operating time" in simple mastoidectomy was lengthened by the use of the endaural route, principally because of the time spent in creating the "mobile, endaural, membranous window." However, the difference in time was shortened as the dexterity of the surgeon increased. We found no essential difference in operating time in the performance of radical mastoidectomy by the endaural route; perhaps the time may be shortened somewhat by obviating the necessity of creating a plastic flap. Certainly the more adequate surgical exposure and the clearness of anatomic detail made the task of the surgeon more enjoyable. Furthermore, it was apparent to us that the surgical treatment of the middle ear was handled with much greater facility than by the postaural approach. It must also be stated that in our attempt to follow Lempert's technic for radical mastoidectomy a much more thorough piece of work was done in the middle ear than was our custom in the past by the postaural approach.

3. Postoperative care and convalescence were not as difficult as we anticipated. It is our belief that there is less discomfort associated with the postoperative care of patients on whom endaural mastoidectomy has been done. Only 2 patients showed any evidence of resentment to the dressings, and these were both children 2 years of age. It is more than probable that these children would have strongly objected to the postaural dressings. None of our older patients on whom mastoidectomy was done experienced any considerable discomfort. None of these patients had myositis, and most of them were out of bed on the fifth to the seventh postoperative day.

These mastoidectomized patients remained in the hospital longer by several days than the patients operated on by the postaural method but only because they were intentionally kept in the hospital for purposes of investigation.

4. In only 1 of the 16 patients who had mastoidectomy was a complication noted. This consisted of a small abscess in the soft tissues just below the mastoid tip, resulting from perforation of the badly diseased mastoid tip by the curet. We wondered how this would drain through the endaural window. After several days, however, the abscess healed and disappeared spontaneously without incision.

Wishart stressed the possibility of an introduction of infection into the temporal region, of perichondritis of the auricle and of constriction of the external meatus. We did not observe any of these complications in our cases.

5. As to results, all our patients on whom simple mastoidectomy was done have dry ears except 1. In this patient, aged 2 years, the middle ear was dry at the end of two weeks, but mild nasopharyngitis developed together with a slight mucopurulent discharge from the middle ear. The adenoids were removed from the patient on the date of the writing of this report.

The 3 patients on whom the modified radical mastoidectomy was performed have greatly benefited by this ingenious operation. All have dry ears, good epidermalization of the mastoid cavity and a striking freedom from granulations.

The results in the 5 patients on whom radical mastoidectomy was performed are encouraging. Three have dry ears with clean radically exenterated cavities. The fourth, who comes from a distance, has failed to return to the outpatient department for check-up. When last seen he had a slight aural discharge. On this patient, a simple and a radical postaural mastoidectomy had been done without good results. The fifth patient is still convalescing at the hospital. Thus far he is doing remarkably well.

As stated, we have performed the endaural fenestration operation for otosclerosis on 7 patients. These patients were selected for the operation only after meticulous study according to the indications as presented by Lempert. As we performed our first operation by this technic only three months ago, we are obviously in no position to pass final judgment on the results. The response thus far in 5 of the patients who have been discharged from the hospital is striking and most encouraging. The other 2 patients are still hospitalized. A further report on the results will be presented in the future.

SUMMARY

The endaural approach for simple mastoidectomy appears to be entirely adequate. Certain features of the method have been presented to indicate a distinct advantage over the postaural route, especially the greater freedom from deformity and the lesser postoperative discomfort. The patients prefer this method.

It is our belief that the endaural approach is the one of choice for radical and modified radical mastoidectomy.

We should not consider any approach other than the endaural one for the fenestration operation.

CLINICAL SIGNIFICANCE OF AURAL POLYPS

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The histologic examination of tumor-like formations in the external auditory canal, frequently observed in association with chronic suppurative otitis media, may yield information of diagnostic, prognostic and corroborative clinical importance. It is well recognized that neoplastic formations such as carcinoma, specific granulomas associated with tuberculosis and syphilis, and manifestations of metabolic disorders may be combined with chronic suppuration of the middle ear. These groups will be discussed briefly with emphasis on certain points of clinicopathologic interest. The main purpose of this communication is to encourage a renewal of interest in the benign neoplastic formations and perhaps a new interpretation of their clinical significance.

CARCINOMA

Malignant neoplasms affecting the middle ear are relatively frequent. The literature shows a paucity of material on the subject, and even any reference there to "primary" carcinoma of the middle ear requires explanation. Proof of the occurrence of such a neoplasm is most difficult. Notably do these neoplastic responses occur in the presence of a marginal perforation of the tympanic membrane in cases of chronic suppurative otitis media, often associated with cholesteatoma. Some authors [1] have commented on this type of suppurative process as having but a casual relationship, but it is of the greatest importance because of the possible invasion of the middle ear by epidermis from the canal and the tympanic membrane. It would be most hazardous to venture a diagnosis in a case of this kind, especially after the tumor has invaded the mastoid or the internal ear and even extensively involved the external ear. It must also be borne in mind that such neoplasms as invade the middle ear and its surroundings may have their origin from the eustachian tube and the pharynx (usually adenocarcinoma) or from the neck, to say nothing of the rarer instances of metastatic origin.

Any neoplastic formation in the canal or the middle ear, whether it evidences a tendency to bleed or not, should be subjected to routine

From the Department of Laryngology, Rhinology and Otology, University of Illinois College of Medicine.

1. Schall, L. A.: Neoplasms Involving the Middle Ear, Arch. Otolaryng. **22**:548 (Nov.) 1935.

biopsy (fig. 1). Recently, in a six week period 5 consecutive cases of carcinoma of the ear presented themselves. All the patients were women. One, 25 years old, had had suppuration of the ear since she was 2 years of age, but the other 4 gave no history of chronic disease of the middle ear. One of these had a malignant process superimposed on a benign tumor of the canal (fibroangioma).

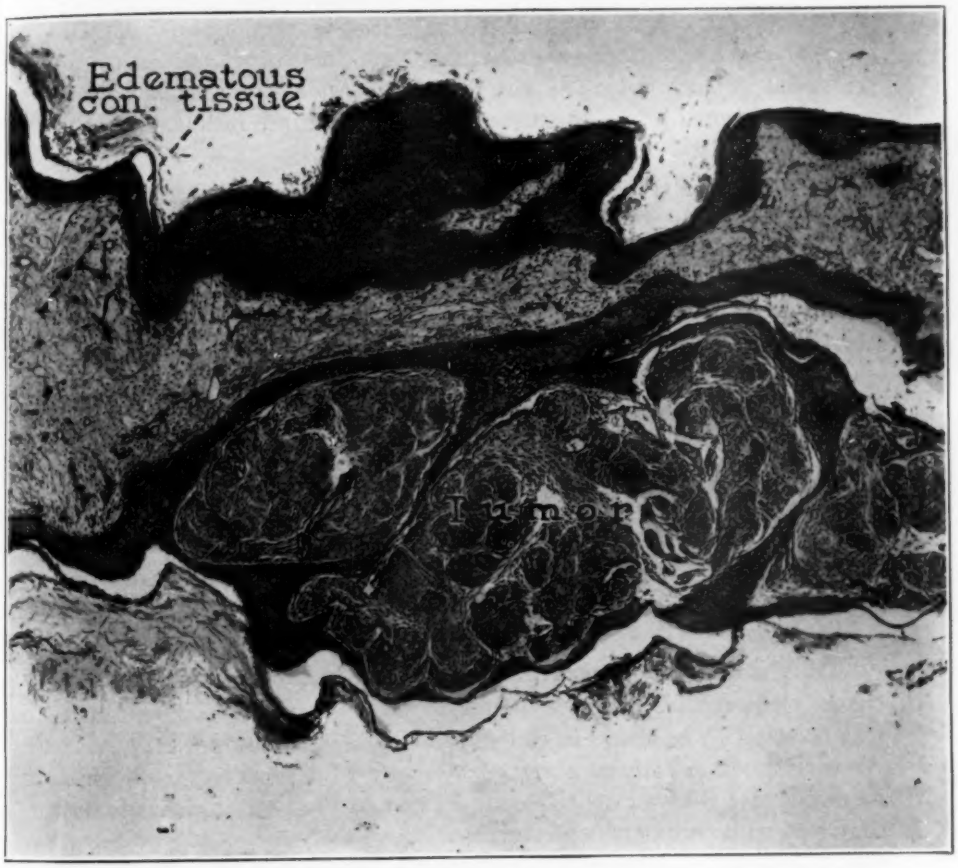

Fig. 1.—Photomicrograph of a polyp removed from a woman aged 47, who gave no history of suppuration. The mass was grossly grayish and did not bleed excessively on removal. It was located mesial to the isthmus of the canal. This polyp was sectioned serially. Study reveals the possible unsuccessful biopsy that would have resulted had only a part of the mass been sectioned. There is observed an encapsulation of the tumor within the tissue of the polyp, also an occasional confluence of cells, which may be interpreted as tumor cells, lying within a lymph vessel and possibly originating from endothelium.

In addition to revealing the type of pathologic change present, I have been able in numerous instances to clarify associated types of involvement, notably those of nerves in the region of the jugular foramen. Such

syndromes have been produced as to form combinations of neural paralyses, which are described in the literature under a confusing array of proper names.[2] In the cases of my series in which the origin of the tumor was probably in the ear, the seventh, ninth and tenth cranial nerves were affected in 3 instances and the seventh in 2; in 4 cases neural paralysis was not presented. Three patients had carcinoma invading the middle and the external ear from the adjacent structures.

Routine histologic study of tissue (serially sectioned) from the external canal will clarify the diagnosis sufficiently early to insure an occasional cure by adequate surgical intervention (radical mastoidectomy) augmented by the employment of electrosurgical technic and postoperative irradiation.

XANTHOMATOSIS

In a previously published paper [3] my associates and I pointed out the great incidence of aural manifestations of lipoid granulomatosis of the skull. Aural suppuration occurs in 50 per cent of the cases, so that the histologic evaluation of a granulomatous mass in the external auditory canal will prevent an unnecessary and perhaps ill advised surgical attack on the mastoid process. A histologic study of such tissue reveals a reticulated structure in large part made up of cells with pale granular cytoplasm and rounded or irregular pale homogeneous nuclei, some of which display indistinct nucleoli. The tissue is also infiltrated by eosinophils and to a lesser extent by neutrophils. Many small blood vessels prevail, and numerous mitotic figures may be present. A similar process in other parts of the skull and in the bones of the body may be revealed by roentgen study.

TUBERCULOSIS

There is considerable controversy as to what constitutes typical tuberculous otitis media, for even the characteristic insidious painless onset may occasionally be absent in certain acute or rapid forms of this disease.[4] An early finding, perhaps observed by many who, however, overlooked its origin, is a thick camphor ice appearance of the tympanic membrane, combined with peripheral vascular injection, in the presence of a profound loss of hearing. Multiple perforations likewise occupy an unwarranted controversial position in otology, since they are observable only in routine otologic practice in a sanatorium which houses patients with early pulmonary manifestations. In the usual course of otologic practice, patients

2. Burger, H.: Einteilung und Nomenklature der "assozierten Stimmbandlähmungen," Acta oto-laryng. **19**:389, 1934.

3. Lederer, F. L.; Poncher, H. G., and Fabricant, N. D.: Aural Manifestations of Lipoid Granulomatosis (Xanthomatosis) of the Skull, Arch. Otolaryng. **21**:27 (Jan.) 1935.

4. Lederer, F. L.: Diseases of the Ear, Nose and Throat, ed. 2, Philadelphia, F. A. Davis Company, 1939, p. 156.

with tuberculous otitis media are seen when the disease is in a later stage and coalescence of the numerous small perforations into one large perforation has occurred.

Aside from a careful analysis of a patient's aural history, histologic study of the granulation tissue from the middle ear may give the most conclusive evidence for the diagnosis of this type of suppuration. This tissue is not in a true sense a new growth and may present all of the characteristics of a tuberculous granuloma (fig. 2). There are two types, fibrotuberculoma (true) and granulotuberculoma (false). The former consists mainly of connective tissue elements resembling fibroma or papil-

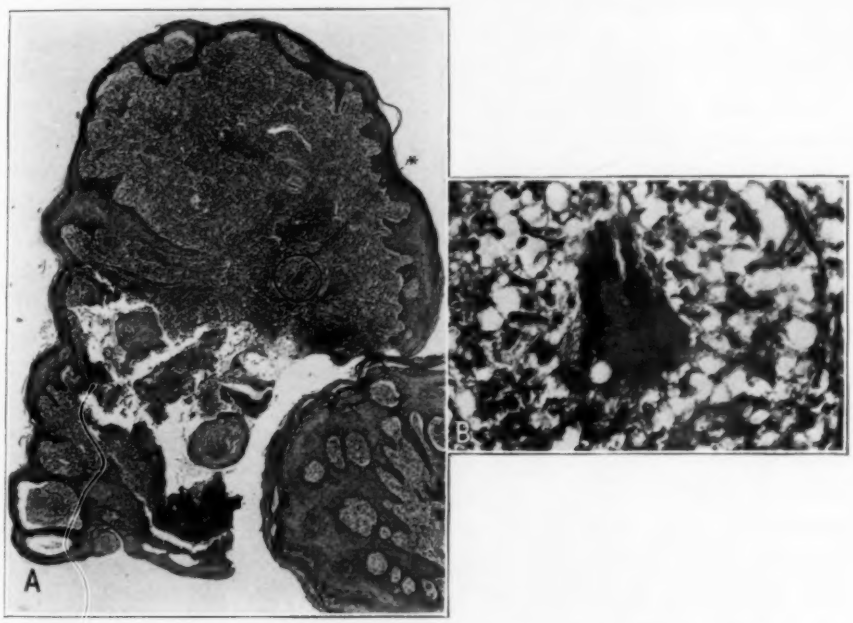

Fig. 2.—Photomicrographs of an aural polyp removed from a patient with tuberculous otitis media. This tissue is not in the true sense a new growth, but its histologic study, correlated with the clinical history, may reveal the diagnosis. The giant cell formation indicated by the encircled area is seen in higher magnification in *B* (× 650).

loma or it may be a typical tuberculous granuloma with characteristic formation of tubercles. The granulotuberculoma possibly represents a healing process resulting from a caseous breaking down of tubercles or the formation of tuberculous granulations in the deeper parts, to become the basis of a papilla-like granulation which breaks through the epithelium, depriving it of its nutrition. The presence of giant cells without tubercles is only presumptive evidence if associated with the clinical picture of tuberculous otitis media.

The indications for medical and surgical management are similar to those which have served as a guide in the care of other types of chronic infection of the middle ear and mastoid. Contrary to preconceived ideas which have been traditionally handed down from otologists of past decades, tuberculous otitis media may heal spontaneously or respond creditably to medical management or to surgical intervention when indications for these forms of treatment are present.

SYPHILIS

A confusing picture is occasionally presented because of a similarity in onset and findings between syphilis and tuberculosis of the middle ear. The presence of a granuloma with giant cells will not in itself indicate a diagnosis of syphilis. Some pathologists believe that as long as one cannot demonstrate spirochetes in the middle ear a syphilitic suppuration per se cannot be definitely spoken of. As a rule, too, spirochetes do not produce a suppurative process. So despite the presence of gummatous lesions elsewhere in the body, it is difficult to rule out disease of the middle ear due to pathogenic organisms. In the experience of some otologists, the incidence of positive serologic findings in cases of pulmonary tuberculosis as encountered in sanatoriums is high and frequently such findings complicate the diagnosis. At the outset of the suppuration the tympanic membrane presents multiple perforations with small pale granulations surrounding the openings. In my experience this condition has been frequently accompanied by local gummatous destruction in the neck and mastoid process and occasionally in the pharynx. The diagnosis of syphilis of the middle ear cannot be made with certainty as long as spirochetes cannot be demonstrated. Moreover, spirochetes seldom produce a suppurative process, so that it is possible that other organisms are responsible for the changes produced.

It is clear that the medical management of such conditions is of first importance, surgical intervention being reserved for releasing pus under pressure or for the removal of bone in a process to be likened to osteomyelitis.

CHOLESTEATOMA

Cholesteatomatous invasion of the middle ear involved in chronic suppuration is not uncommon. The genesis and pathologic aspects of such a disease are well known and its treatment by surgical means more or less universally agreed on. For those whose experience causes them to condone an expectant attitude in such cases, an occasional observation of acute exacerbations and the finding post mortem of old, well walled-off brain abscesses will serve to alter a fixed policy of expectant, so-called conservative management in similar cases. In addition to revealing the

presence of cholesteatomatous formations within the polyp (fig. 3), the histologic picture in a number of cases gives evidence of acute exacerbation of granulation masses. This point will be further elucidated in the consideration to follow.

Fig. 3.—Photomicrograph of a cholesteatoma. What appear to be lipoid deposits are in reality cholesteatomatous deposits (*Ch*) within the granulation tissue.

BENIGN AURAL POLYPS

The appearance of polyps in an ear affected with suppurative otitis media is a sign of chronicity. The purulent process cannot be expected to heal *ad integrum* except in an infant, in whom the presence of a polyp

does not carry the same significance as in an adult. In a typical case of chronic suppuration accompanied by the formation of polyps in an adult, complete return to normal healing and hearing is not expected. This is of importance, since the literature contains reports that polyps may occur in association with an acute suppurative process in an adult.

According to the previous conception of otologists, a granulation polyp evidences a suppuration of bone. It is well known that not all suppurations of bone per se necessitate surgical intervention. This indication is dependent on the stage, viz., (*a*) quiet (not exacerbated); (*b*) showing a tendency toward healing, at least for a time, and (*c*) acutely exacerbated. The study of an aural polyp may reveal the different stages of bone suppuration. By means of such an examination the otologist has an excellent opportunity to determine the index of the suppuration at the time of the extraction of the polyp. It must be borne in mind that the extraction of a polyp during an acute exacerbation is dangerous, because the procedure may make manifest a latent intracranial complication. For this reason the extraction of an aural polyp should be performed by an otologist who is in a position also to cope with an eventual complication.

Otologic literature contains numerous references to the development of aural polyps from the walls of the middle ear, from the eustachian tube, the ossicles and from the walls of the pneumatic spaces in association with bone disease. They occur singly or in multiples and vary from pinhead size to such proportions that they occlude the entire canal and present themselves in the external auditory meatus. Their consistency varies from extreme softness to hardness, and they appear intensely red or pale, lobulated and mulberry-like or small and oval.[5]

Two groups of aural polyps have heretofore been considered, viz., mucous membrane polyps and granulations. Some question has existed as to whether the different forms of polyp encountered, such as the granulation, fibroma, myxoma and transitional forms, originated as such or represented merely transformations of mucous membrane polyps. These various tumors have been considered to be of inflammatory origin; they consist of a mixture of polymorphonuclear leukocytes, plasma, mast and giant cells and fibroblasts, which lie in a fibrous stroma containing numerous newly formed blood vessels. The surface covering has usually been said to consist of pavement epithelium. The younger structure has been described as containing less elastic tissue but rich in a hyaline homogeneous stroma traversed by a fibrous framework in which are interspersed many round cells and a few spindle cells. The surface of aural polyps has often been described as ulcerated and devoid of epithe-

5. Brühl, G.: Lehrbuch und Atlas der Ohrenheilkunde, ed. 4, Munich, J. F. Lehmann, 1923, p. 445.

lium, or covered by flat, or by cylindric, epithelium, or by both types. The epithelium frequently presents glandlike indentations into the stroma, where glands seldom occur.

Mucous membrane polyps originate from folds in the mucous membrane, which protrude and are covered by the same type of epithelial layer as the lining of the middle ear. Within the polyp the epithelial layer may produce secondary folds of epithelium which is highly cylindric and contains a number of goblet cells. If, in sectioning, these folds lose their connection with the remaining epithelium, they may simulate the histologic picture of a gland.

This is the first possibility suggested by Wittmaack.[6] The second is as follows: It is known that in otitis of infants the exudate may become organized in such a manner that it may be transformed by the blood vessels in the subopenings of the epithelial layer into a granulation plug. When this collection of organized exudate increases in size, it may produce the picture of a polyp. Polyps developed by such a mechanism may have different types of epithelial layer, depending on the epithelium of the middle ear. The connective tissue of these polyps also shows changes which depend on the structure of the mucous membrane of the middle ear. Sometimes a loose network of connective tissue is found, and occasionally it is of a firmer character. Cysts are also to be found. While the polyps of the first group, according to Wittmaack, usually develop in suppurations surrounding the eustachian tube, those of the second group develop in cholesteatomatous processes of the attic; these are the so-called granulation polyps, which consist of granulation tissue and are not covered by a layer of epithelium. The presence of such polyps almost always signifies osseous necrosis. In such cases, when the necrotic tissue is removed the polyp may shrink spontaneously; in other cases, of course, the polyp does not shrink but may be covered by a layer of squamous epithelium, so that in this stage a differentiation between granulation and mucous membrane polyps cannot be made.

The granulation polyp is derived from the subepithelial layer of the mucous membrane and therefore primarily is not covered by epithelium but becomes so secondarily during the course of the disease. Polyps of both types undergo changes in tissue during the course of the otitis media, these alterations being dependent on the stage (duration) of the disease, the length of time that the tissue was subjected to mechanical irritation from caustics, the pressure and discharge and the circulation (stagnation) within the polyp.

These possibilities explain how the tissue may undergo alterations in size, form and structure and why the older otologists have described

6. Wittmaack, K., in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1926, vol. 12, p. 248.

histologic fibroma, myxoma and angioma, although it is apparent from my studies that they are one and the same tissue. The usual division of polyps previously described [7] may be well founded theoretically, but from a practical point of view the argument to be offered against it is that various investigators may obtain different conceptions of the tumor, according to whether the polyp is studied in association with the mucous membrane of the walls of the middle ear or is disassociated from its surroundings and examined separately.

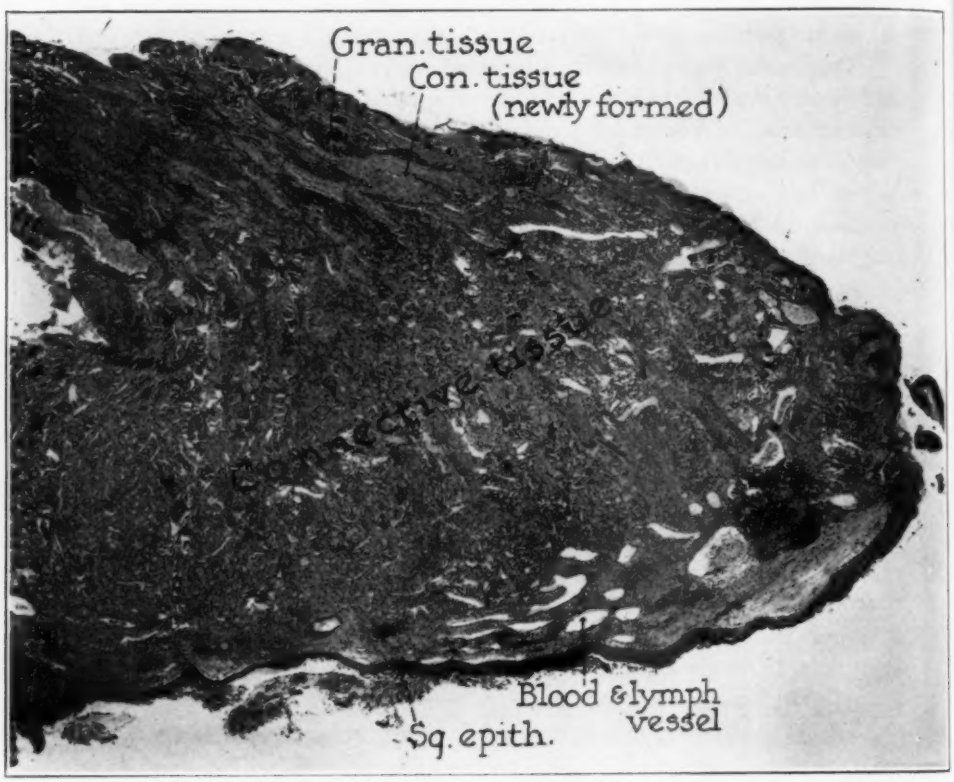

Fig. 4.—Photomicrograph of a granulation polyp. This polyp consists of granulation tissue which contains many mononuclear leukocytes, lymphocytes and plasma cells. Polymorphonuclear leukocytes are sometimes observed to be heaped up within the lumen of the blood vessels but are not of importance within the granulation tissue.

Previous investigators have attempted an analysis of aural polyps on the basis of the plasma cell content. Some have suggested that the

7. Seydell, E.: Ear Polypi in Acute Otitis Media, Ann. Otol., Rhin. & Laryng. **34**:925, 1925.

basis for the formation of polyps and granulations is frequently a disease of the bone and have therefore given a more unfavorable prognosis when there was evidence of this origin than when the study revealed structural characteristics resembling those of fibroma or myxofibroma. Only a small percentage of polyps present such true tumor formation, with

Fig. 5.—Photomicrograph of a granulation polyp. This polyp presents a papillary appearance (*Pap.*).

compact tissue poor in blood vessels, rich in elastic fibers and covered by flat or cylindric stratified squamous epithelium.

This report is based on a study of 59 cases in which polyps were examined in the course of routine laboratory procedure. Thirty-three

of the polyps can readily be divided into four distinct types, and the remainder can be classified as neoplastic and metabolic formations of the kind previously described.

1. Granulation Polyp.—To this group belong 10 polyps in my series of cases. The growths consist of granulation tissue which contains many mononuclear leukocytes, lymphocytes and plasma cells, while poly-

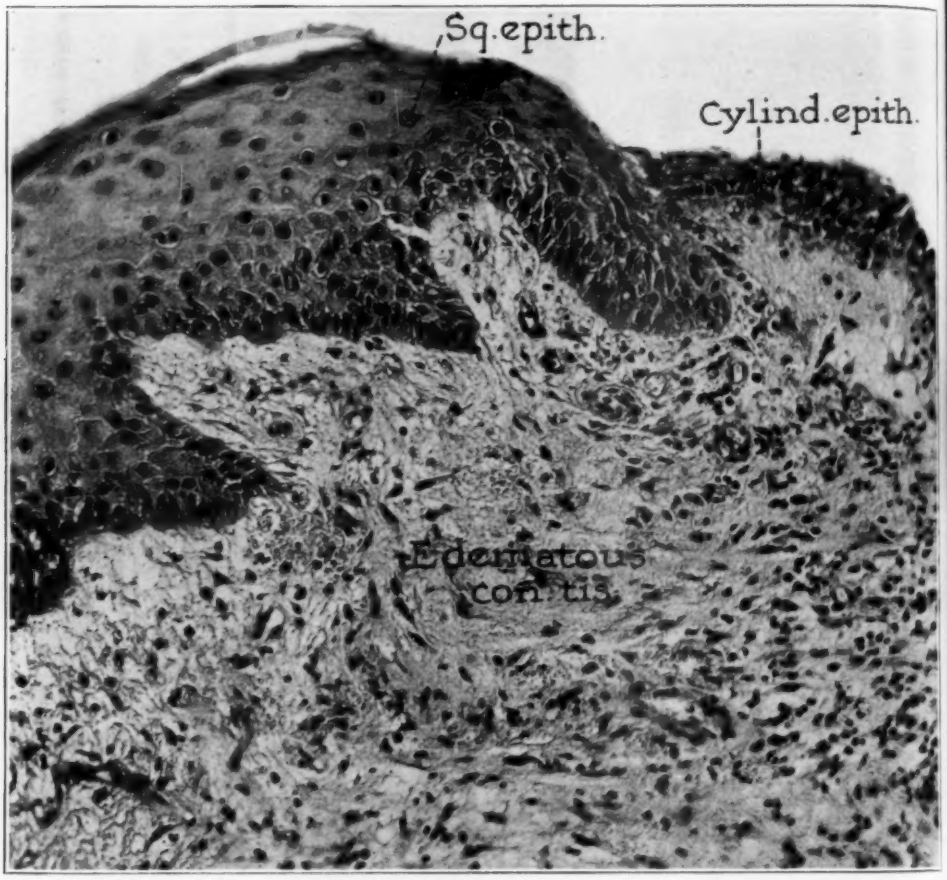

Fig. 6.—Photomicrograph of a granulation polyp. This section illustrates the impasse reached when squamous and cylindric epithelium meet.

morphonuclear leukocytes are sometimes heaped up within the lumens of the blood vessels but, are not of importance within the granulation tissue (fig. 4). There are always fibroblasts to be seen, however, and in some areas their number is so increased that the granulation tissue is changed into a loose edematous connective tissue (fig. 5). Both types of tissue are frequently observed in one polyp, so that the differentiation

between granulation polyps and mucous membrane polyps is not substantiated by this study. At times giant cell–like formations are observed within the granulation tissue.

There may be many or few blood vessels present. The difference in the number is not readily explained. However, when many blood vessels are found, evidence of recent and old hemorrhages (pigment) is observed within the polyp.

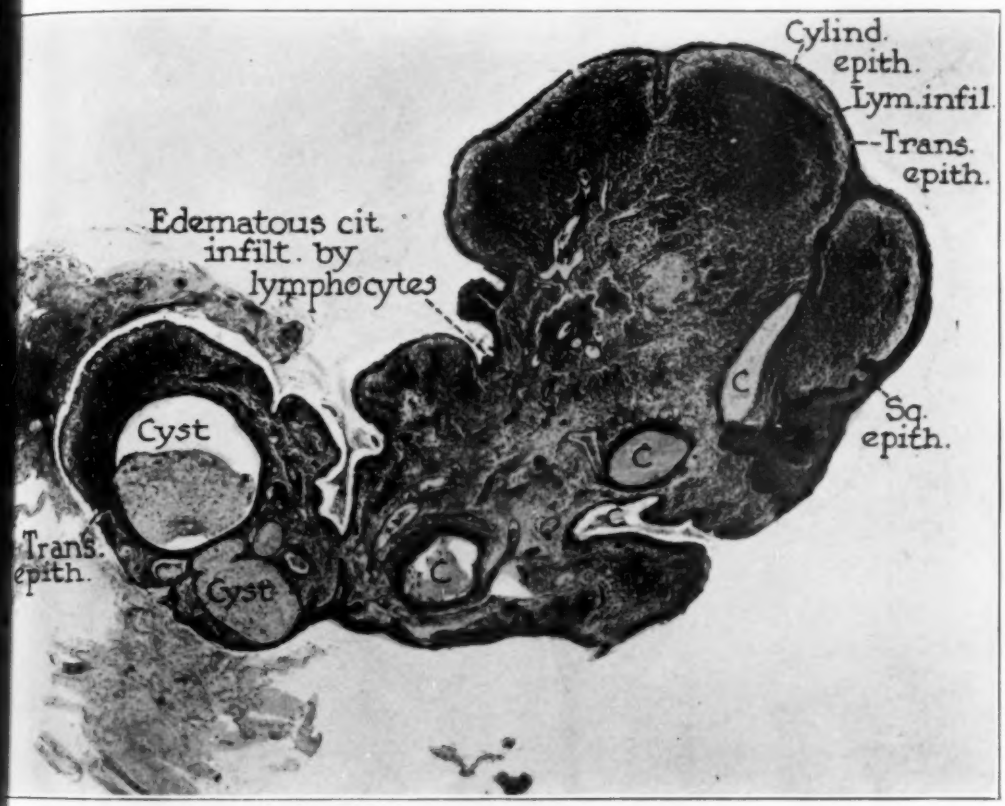

Fig. 7.—Photomicrograph of a granulation polyp greatly infiltrated by lymphocytes and containing numerous cysts. The mechanism of the formation of a cylindrically lined cyst is well illustrated to the right of the section.

The granulation polyp may be covered solely by a layer of hornifying squamous epithelium with deep cones. More often, however, there are layers of different types of epithelium, high cylindric, cubic, transitional and squamous, on the surface of the polyp. Each variety of epithelium is sharply separated from the others (fig. 6). It is of importance that, as was previously mentioned, the deep or shallow folds, or crypts, of the squamous epithelium are found in the granulation tissue (fig. 7). If

the epithelium is in folds, as often occurs, the squamous epithelium ends at the entrance into the fold, the walls of which are lined with cylindric epithelium, with or without goblet cells. The folds are often deep and may in some sections simulate glandlike formations or cysts. These "cysts" may or may not be filled with a thready mass (probably mucus),

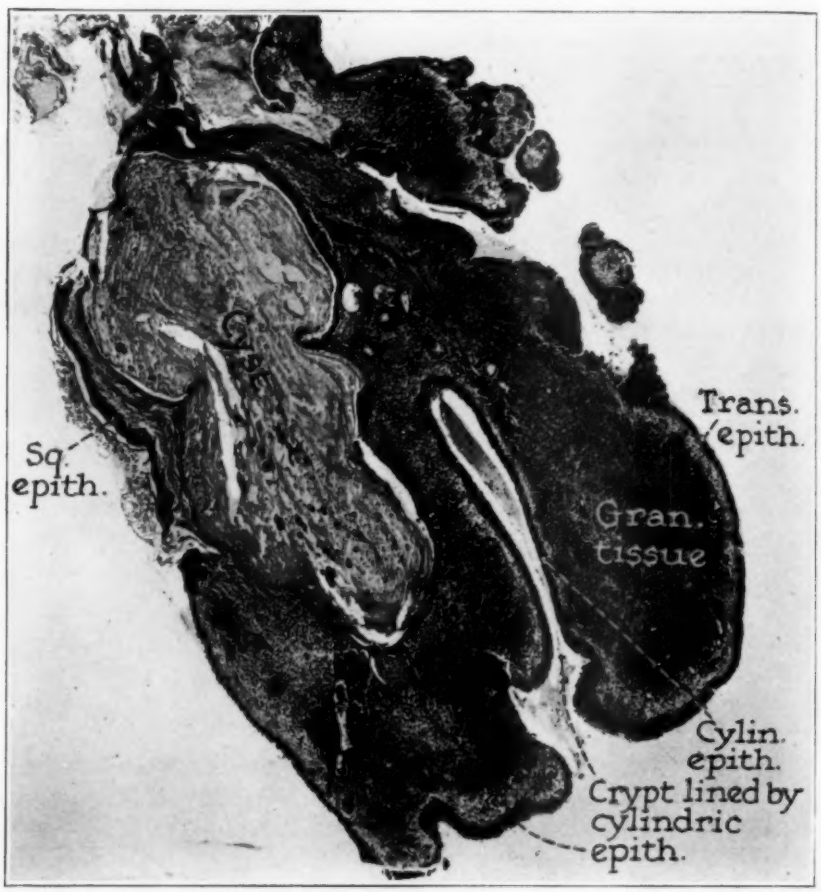

Fig. 8.—Photomicrograph of a granulation polyp presenting a papillary appearance and cyst formation.

which is sometimes so developed that the cylindric epithelium becomes flattened and the cysts dilated (fig. 8).

All the epithelium is permeated by leukocytes, mostly polymorphonuclear, and often both show signs of disintegration. However, an accumulation of polymorphonuclear leukocytes does not usually occur under squamous epithelium. On the contrary, there may be a greater

number of polymorphonuclear leukocytes under cylindric epithelium, especially where it produces folds, glandlike formations and cysts.

2. Connective Tissue Polyp.—To this group belong 4 polyps in my series. These growths consist of a more or less dense connective tissue with so many blood and lymph vessels that occasionally one gets the

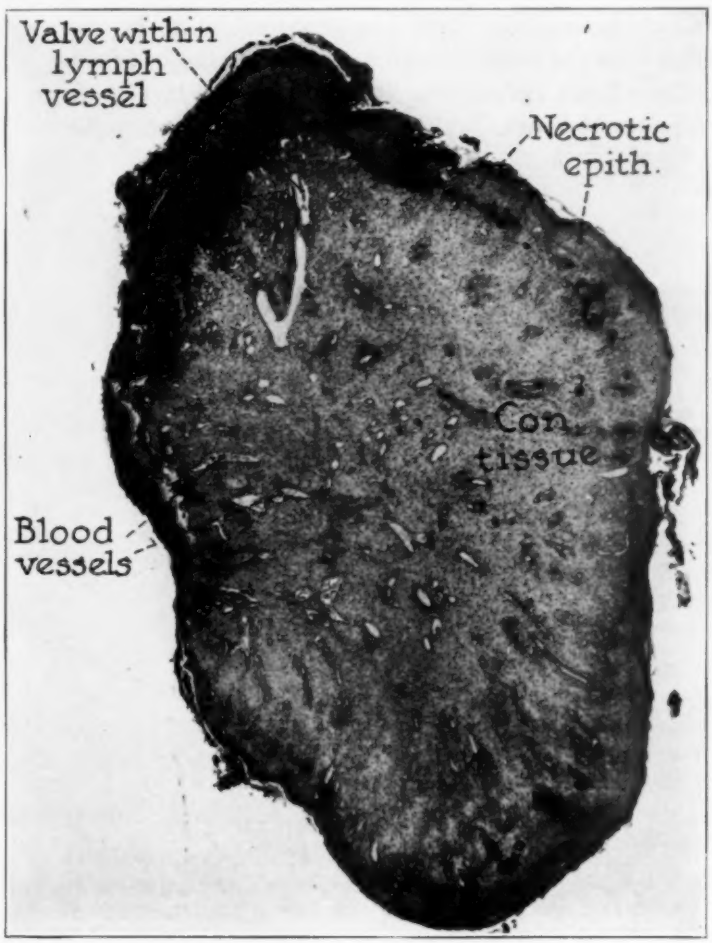

Fig. 9.—Photomicrograph of a connective tissue polyp containing many newly formed vessels.

impression of an angioma or a fibroangioma. In some of the vessels even valves are observed (fig. 9). There is always an accumulation of cells around the vessels. These cells are mainly mononuclear leukocytes, with a few polymorphonuclear leukocytes, fibroblasts, histiocytes (often loaded with blood pigment) and fibrocytes.

The majority of these growths are covered by squamous epithelium with deep cones or a simple layer of flat cells, which simulates the picture of an endothelium. In a case in which the squamous epithelium had become necrotic, there were observed many disintegrated leukocytes and bacteria. There were also dilated blood vessels and evidence of recent hemorrhages surrounding the necrotic area. It is most probable that the necrosis of the epithelial layer was produced by an infection entering the polyp from the external canal and not from the middle ear.

3. Granulation Polyp, Acutely Exacerbated.—In this group belong 13 polyps in my series. These consist of granulation tissue, as do the

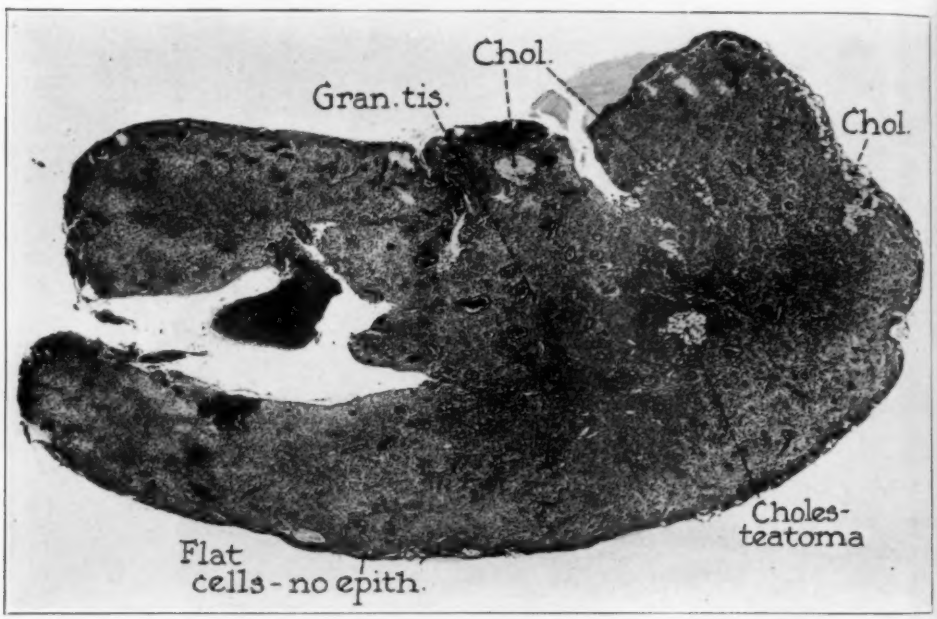

Fig. 10.—Photomicrograph of a granulation polyp in an acute exacerbation of a chronic, cholesteatomatous process.

polyps described under group 1. In this granulation tissue there are sometimes giant cell–like formations, which appear like vessel buds and frequently evidence recent and old hemorrhages. The evidence of old hemorrhages can readily be recognized by the pigment, which in some cases is so well developed that the walls of the capillaries are impregnated by it.

The surface of these polyps usually reveals no epithelium at all or a necrotic squamous epithelium which is permeated by many polymorphonuclear leukocytes and covered by bacteria (fig. 10). In many cases

there are some remains of normal squamous epithelium and seldom the remains of cylindric epithelium.

Up to this point these polyps do not differ markedly from those described under group 1, the great difference being the appearance of a great number of polymorphonuclear leukocytes. These are accumulated particularly near the surface of the polyp, where they may form real abscesses, while they are less frequently seen within the center of the polyps (fig. 11). Such an arrangement suggests that the leukocytes have invaded the polyp from the surface. This is not the case, for it

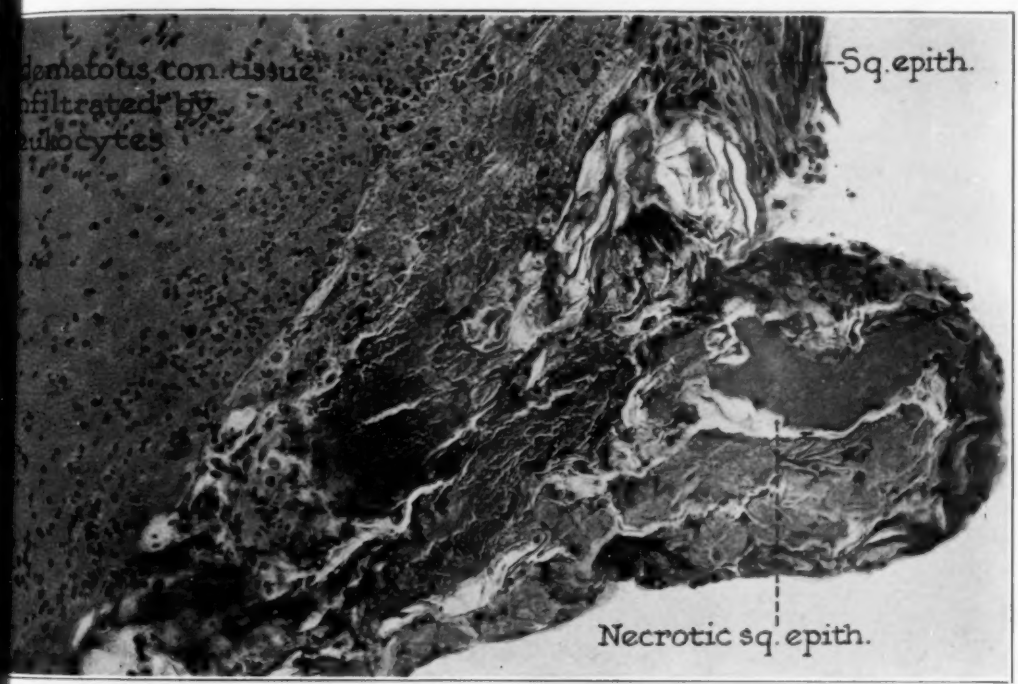

Fig. 11.—Photomicrograph of an acutely exacerbated granulation polyp. The leukocytes are seen migrating from the center of the polyp to the surface epithelium, which is being lifted up by the necrotic process.

can be definitely observed that they enter the polyp within the blood vessels which originate from the mucous membrane of the middle ear and that they permeate the walls of the blood vessels and wander from the center to the surface of the polyp, where they penetrate the necrotic epithelial layer to form the purulent secretion within the external canal (fig. 12). This is the characteristic picture of an acutely exacerbated polyp.

Of particular interest and import is the stage of acute exacerbation, in which surgical intervention is usually indicated. It is true that there are other signs, but in cases in which the polyp is obturating the canal the mucous membrane of the middle ear cannot be visualized. Histologic

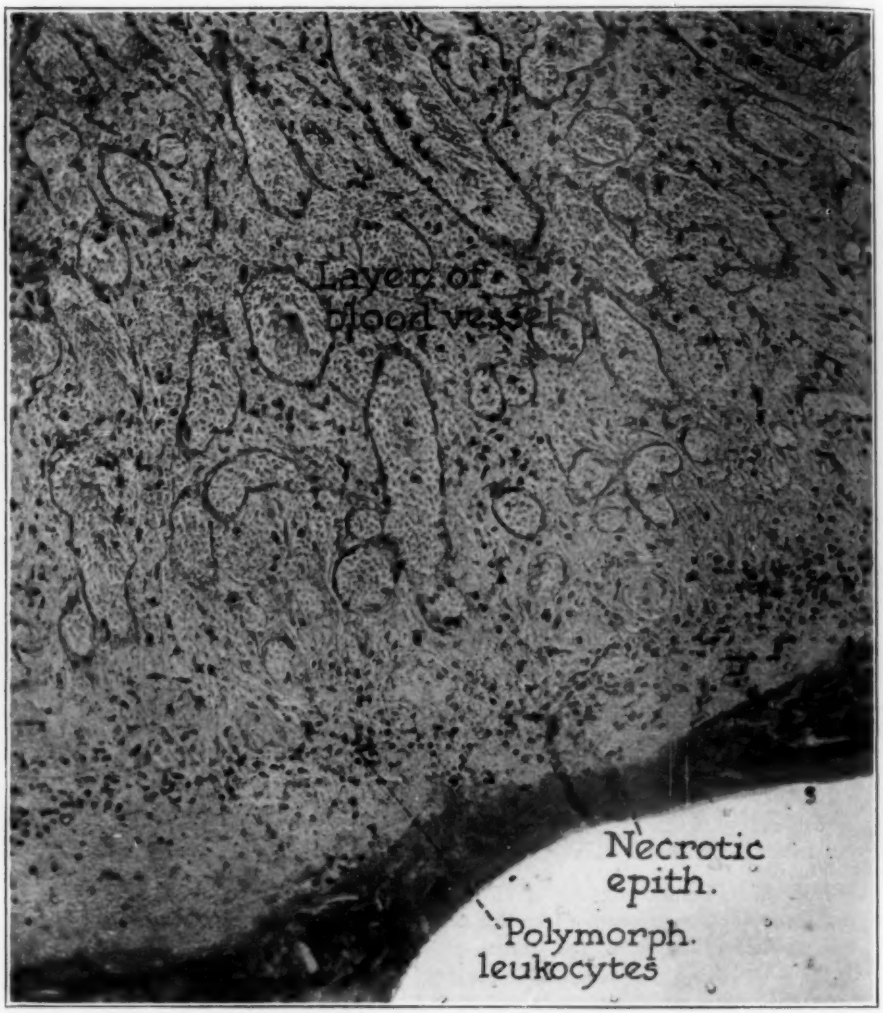

Fig. 12.—Photomicrograph of an acutely exacerbated granulation polyp. The area of necrosis is filled with pigment and disintegrated leukocytes.

study of the polyp therefore permits a conclusion regarding the necessity for surgical procedure.

There is no doubt that the polyps mentioned under groups 1, 2 and 3 show only different stages of one disease, viz., chronic suppuration of the middle ear. Several points are apparent: Group 1. These polyps

show essentially the same structure as the mucous membrane of the middle ear in a case of severe chronic otitis media. Group 2. These show essentially the same structure as the mucous membrane of the middle ear in a case of chronic otitis media in which the membrane has a tendency to dry up. Group 3. These show essentially the same structure as the mucous membrane of the middle ear in a case of acute exacerbation of chronic otitis media.

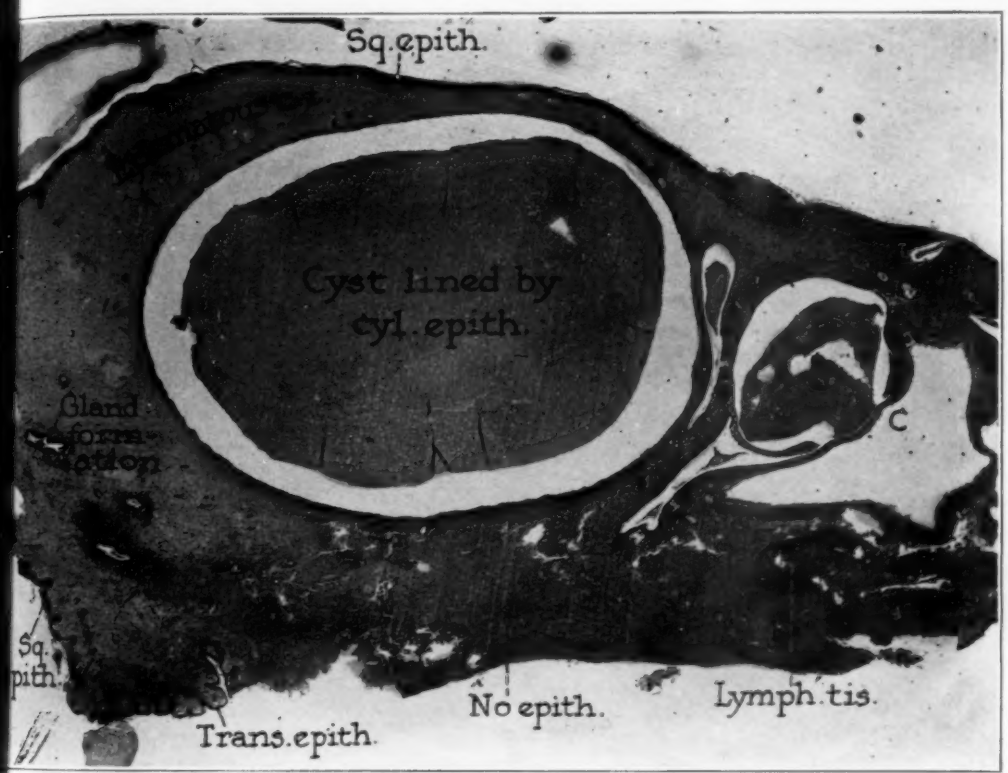

Fig. 13.—Photomicrograph of a lymphatic polyp containing a large and smaller cysts (*c*). Within the edematous connective tissue (*c. t.*) definite gland formation and lymphatic tissue are seen.

4. Lymphatic Polyp.—To this group belong 5 polyps in my series. These structures definitely differ from those described under groups 1, 2 and 3. It is true that they also consist of granulation tissue with a few polymorphonuclear leukocytes. This granulation tissue, however, evidences a definite transition into an edematous connective tissue, so that in large areas these polyps resemble nasal polyps (fig. 13).

Two characteristics should be kept in mind: 1. While in polyps of groups 1, 2 and 3 squamous epithelium prevails, in lymphatic polyps cylindric epithelium is definitely preponderant. Therefore, the greatest part of each of these polyps is covered by cylindric epithelium with many goblet cells, deep folds, glandlike formations and partly filled (with mucus?) cysts. 2. The tissue of the entire polyp is infiltrated by lymphocytes, which are particularly accumulated under the cylindric epithelium while they are scanty within the center of the polyp. Under the cylindric

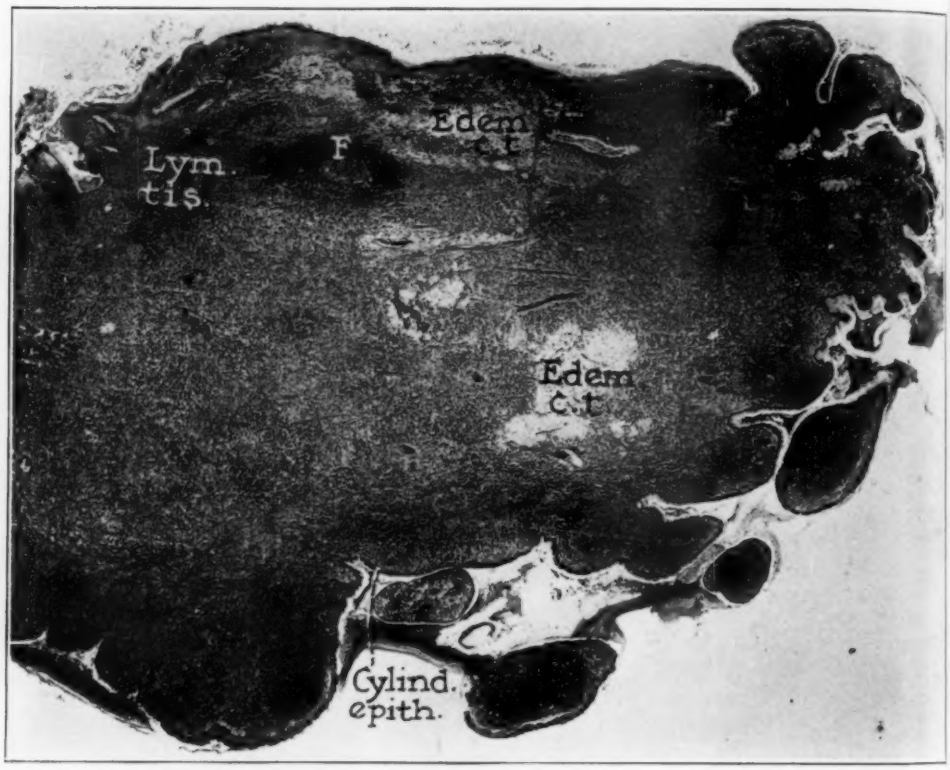

Fig. 14.—Photomicrograph of a lymphatic polyp. A follicle (*F*) contains a germinative center. The cylindric epithelium contains many goblet cells.

epithelium the lymphocytes may often produce follicles with germinative centers, so that the tissue resembles lymphatic tissue, which explains the term "lymphatic polyp" (fig. 14).

Inasmuch as a similar tissue is found only within the eustachian tube and the hypotympanum, it is probable that lymphatic polyps originate in these parts of the middle ear. This form of polyp characterizes suppuration of the mucous membrane, and therefore surgical intervention per se is not indicated. Such tissue responds admirably to radium therapy.

COMMENT

The examination of material in the series of cases in my clinic consisted of a study of polyps, disassociated from knowledge of the condition of the mucous membrane of the middle ear. From such an investigation one can state that generally examination of the polyp will throw some light on the condition of the mucous membrane of the middle ear.

I cannot agree with divisions into polyps and granulations, as both are incidental possibilities and therefore one can use "aural polyps" only as a general term and from such a study draw certain conclusions as to the condition of the mucous membrane of the middle ear. It is possible to conclude: (1) that the tissue may evidence neoplastic and metabolic disease, such as carcinoma, cholesteatoma, tuberculosis, syphilis and xanthomatosis, (2) that all stages of suppuration, and particularly an acute exacerbation of chronic otitis media, may be diagnosed and the condition of the lining mucosa of the middle ear possibly otherwise evaluated from a histologic study of a polyp which occludes the middle ear, by virtue of the polymorphonuclear leukocytic infiltration.

There is no doubt that the division of polyps into two groups, as Wittmaack postulated it, is theoretically acceptable, but its practical clinical value is questionable. According to Wittmaack, polyps change their structure, depending on the stage of suppuration of the middle ear, and it is therefore difficult to differentiate in general between the two types of polyps. The localization of the polyps, likewise, does not mean much. Inasmuch as it is not practical to accept Wittmaack's classification in the case of large aural polyps whose origin one cannot determine, it is far better to draw basic conclusions as to the condition of the mucosa of the middle ear from examination of the polyp.

The division into groups as presented in this communication is only the beginning of further research, for up to this time polyps have been studied from a purely histologic point of view. From now on, such a study must be associated with that of the history, clinical course and findings. It is agreed that the division into these groups is probably schematic and that subsequent changes may be required, but it is felt that it outlines a course for future investigation.

Case Reports

ACUTE MASTOIDITIS COMPLICATED BY SCHÜLLER-CHRISTIAN DISEASE

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The unusual combination of a common pyogenic infection and a rare and infrequent metabolic disturbance of the bones of the skull has prompted the following report of a case of Schüller-Christian disease.

The basis on which acute streptococcic otitis media and mastoiditis developed in a child 3 years old was felt to be impaired nutrition of the temporal bone. The case presented the usual problem of a simple mastoidectomy. After the evacuation of the postaural abscess, the mastoid cortex was found to be a sequestrum, which was easily removed, with most of the mastoid cells. This was thought to be unusual, and after the completion of the operation, roentgenograms of the skull were taken, which revealed the presence of Schüller-Christian disease involving both sides of the skull, as shown in figure 1. The opposite ear had not shown any evidence of an acute infection, even though roentgenograms revealed a similar lipoid necrosis of the opposite temporal bone.

Schüller-Christian disease is a disturbance of fat metabolism, the cause of which is unknown. Pathologically it consists of a lipoid cell hyperplasia within the reticuloendothelial system, a system which is present in many parts of the body, including the area between the inner and outer tables of the skull. As this abnormal deposit of fat occurs in the reticuloendothelial system the calcium softens, the blood supply is impaired and dissolution of bone takes place (Rowland[1]). As a result, there occur within the skull punched-out areas, which are quite characteristic of this condition as studied in roentgenograms. "Lipoidosis," "lipoid granulomatosis" and "xanthomatosis" are all terms expressing essentially the same pathologic condition, although the latter is much the broadest term and covers this abnormal disease of fat metabolism in other parts of the body.

Microscopic sections in the cases reported have shown large, pale round or oval cells containing fat droplets. These are called foam cells (xanthoma cells). They are surrounded by plasma cells, leukocytes and fibroblasts, which comprise lipoid granulation tissue. The foam cell is thought to be a reticuloendothelial cell which has undergone a change because of altered lipoid or cholesterol metabolism (Thannhauser[1]).

REPORT OF A CASE

J. J., a girl aged 3, entered Children's Hospital Jan. 11, 1939, with the following history: Delivery had been normal at full term. She had been apparently well until one year before admission, when an adenoidectomy had been performed. She

1. Thannhauser, S. J., in Christian, H. A.: Oxford Medicine, New York, Oxford University Press, 1940, vol. 4, p. 214.

had been subject to frequent colds and bronchitis and had gradually become a mouth breather. Shortly after the adenoidectomy her thirst had increased rapidly and excessive urination had begun. This condition had grown progressively worse until three weeks before admission, when a tonsillectomy and a second adenoidectomy, for the mouth breathing, had been performed. Two weeks later the left ear had begun discharging, and a postaural swelling had appeared over the left mastoid, which was fluctuant and tender. The temperature was 103 F., the pulse rate 116 and the respirations normal. There had been loss of appetite and a slight loss in weight.

On admission, the child appeared emaciated, poorly nourished and pale, and the skin was rough and dry. Over the left mastoid bone was a postaural swelling.

The pharynx was moderately injected, and neither tonsillar fossa had completely recovered from the recent tonsillectomy. The chest was asthenic in appearance, with prominence of the ribs, and bronchovesicular breathing was heard throughout. The abdominal veins were prominent, and the liver was enlarged to a point below

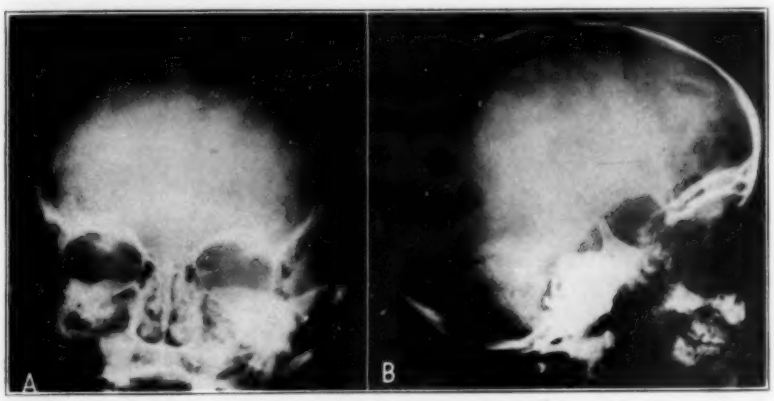

Fig. 1.—*A,* anteroposterior view of the skull, showing lipoid deposits in the bones of both orbits. *B,* lateral view of the skull. A clearly demarcated area appears in the frontal and parietal bones on each side. The sella turcica is greatly enlarged. Deposits of lipoid cells are seen over the left mastoid bone, with skin clips in situ.

the costal margin. The spleen was not palpable, but there was enlargement of inguinal, axillary and cervical glands bilaterally.

Laboratory Findings.—The white blood cells numbered 16,200 per cubic millimeter; the differential count was normal. The red blood cells numbered 3,600,000 per cubic millimeter. During fasting the blood sugar was 84 mg.; one-half hour later it was 151 mg., and one hour later, 166 mg. The blood cholesterol on two occasions was 143 and 138 mg. per hundred cubic centimeters. Examination of the urine showed no abnormalities except a low specific gravity and a total output of between 3,000 and 4,500 cc. daily.

Operation (January 11).—The routine incision was made over the mastoid and a postaural abscess evacuated. The cortex of the mastoid was moth-eaten, so that the landmarks were obscured. In the region of the tegmen antri lay a sequestrum, which on removal proved to be the inner table of the skull. Granulations overlay the dura. Since it was realized that this was an abnormal condition and that the acute infection was relieved, the wound was closed and a drain inserted.

Roentgenologic Report (Dr. F. B. Stephenson).—"There is evidence of bone absorption or bone replacement involving the body of the sphenoid and the clinoid processes, the roofs of both orbits, the greater wings of the sphenoid and neighboring parts of both frontal bones and the lower portions of the temporal bones extending backward to the mastoid regions. It is more extensive on the right than on the left. The sharp demarcation of the diseased bone is evidence against osteomyelitis. There is also evidence against pressure atrophy. The most likely diagnosis is xanthomatosis. There is no evidence of faulty development of the wrists."

Postoperative Course and Treatment.—The child remained afebrile for several days postoperatively, requesting water every fifteen minutes and urinating as often. The intake of water and the output of urine were about 3,500 cc. and 3,300 cc. respectively. At home the uncontrolled intake of water was 4,000 cc. to 5,000 cc. daily. The child's weight was 25 pounds (11 Kg.). All fat was eliminated from her diet. A series of high voltage roentgen treatments, twelve in all, was

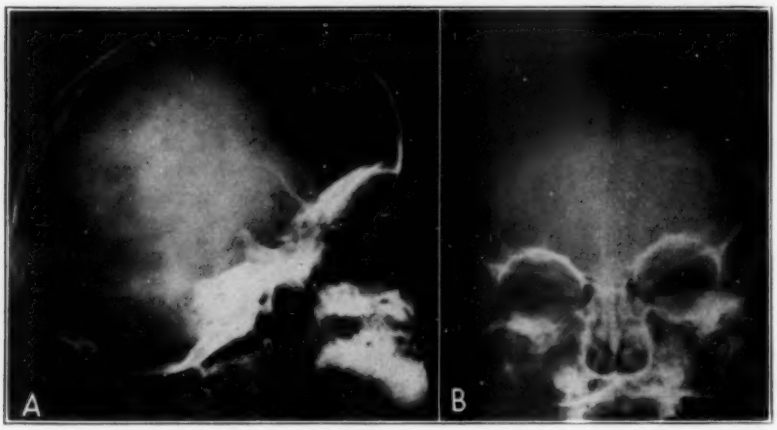

Fig. 2.—*A,* lateral view of the skull, showing complete calcification of the bony orbits and the parietal frontal junction on the right side, following high voltage roentgen therapy. *B,* anteroposterior view of the skull, showing calcification of the floor of the sella turcica and regeneration of the anterior clinoid process following high voltage roentgen therapy.

begun January 25 and terminated February 6. The next few days showed abatement of the diabetes insipidus. The child was not asking for water as often as before and was soon able to go four hours at night and from one to three hours in the daytime without urination. The ratio of intake of water to output of urine improved over a two week period, the output being one third of the intake. The administration of solution of posterior pituitary U. S. P. was purposely omitted, so that it might be determined whether the high voltage roentgen treatment was effective. To improve her weight, fat was put back in her diet, and this addition seemed to have no effect on the blood cholesterol. Epilation of the scalp resulted from the roentgen treatments, and routine roentgenograms of the skull made on February 19 showed no extension of the process and as yet no deposition of calcium in the fat-laden areas. Roentgenograms of the parents and of 2 other children showed no abnormal familial tendencies.

The mastoid wound and the ear continued to discharge until February 26 and at the time of writing are healed and dry. The weight has increased to 27 pounds (12 Kg.). Roentgenograms of the skull taken eighteen months after discharge from the hospital showed recalcification of the xanthomatous areas of both mastoids, of the bony orbits and of the clinoid processes of the sella turcica (fig. 2).

During two years, while the child has been under observation, she has gained in weight, the hair has regrown after complete epilation and only two attacks of otitis media have occurred in the diseased ear, both of short duration. There has been no recurrence of the mastoiditis. Subsequent roentgenograms have shown the defects of the skull in figure 1 to be filled in with new bone after the twelve high voltage roentgen treatments. The exophthalmos, which was very slight in the beginning, has apparently remained the same, and the diabetes insipidus has subsided. From a physical as well as a roentgenologic point of view, the patient can be considered cured, even though acute exacerbations of a chronic otitis media may contiue to occur. These have responded to treatment in the past, and between attacks the ear is dry.

COMMENT

To distinguish Schüller-Christian disease from the other disturbances of lipoid metabolism, it is necessary to have present a triad of physical findings consisting of:

1. Bony defects confined to the skull.
2. Exophthalmos.
3. Diabetes insipidus.

These symptoms were consistently found in the early cases reported by Schüller,[2] of Vienna, and later by Christian,[3] in this country. To Rowland[4] belongs the credit for establishing the pathologic basis for diagnosis. This has opened a new field for investigation, and since his work in 1928 many varieties of lipoid disturbances throughout the body have been discovered.

Several cases of Schüller-Christian disease associated with otitis media have been reported in the literature. In some the disease has been associated with chronic otitis media and in some with mastoiditis. The labyrinth, the petrous pyramid, the facial canal and both parts of the temporal bone have been involved in this disease. The frontal and the parietal regions are the parts of the skull most commonly involved, and the face may be affected, with involvement of the maxilla and loss of the teeth. Rarely are the liver and spleen found to be enlarged.

The cholesterol content of the blood, which is the only laboratory finding of significance, has been reported to be both unusually high and low, with no consistent interpretation. In the case reported here it was within normal limits on two occasions.

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2. Schüller, A.: Wien. med. Wchnschr. **71**:510, 1921.

3. Christian, H. A.: M. Clin. North America **3**:849, 1920.

4. Rowland, R. S.: Xanthomatosis and Reticulo-Endothelial System: Correlation of Unidentified Group of Cases Described as Defects in Membranous Bones, Exophthalmos and Diabetes Insipidus (Christian's Syndrome), Arch. Int. Med. **42**:611 (Nov.) 1928.

The maximum number of patients with this disease have ranged between the ages of 2 and 4 (Dauksys [5]), and there appears to be no familial or racial proclivity. Cases on record indicate that the disease has occurred in Negroes, Jews and Maoris (Stowe [6]) and in members of the same family. Roentgenograms of the parents of the patient whose case is reported here failed to show any involvement in either.

The literature reveals some recoveries as well as deaths, the mortality ranging from 30 to 50 per cent. The majority of authors are in agreement that roentgen therapy is the only treatment of value except when complications occur which in themselves require surgical intervention (postaural abscess).

5. Dauksys, J.: M. Bull. Vet. Admin. **13**:40, 1936.
6. Stowe, W. R.: M. J. Australia, 1927, supp. 5, p. 144.

ATROPHIC RHINITIS WITH OZENA

Etiologic Factors

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CHICAGO

Any evidence which helps to clarify the etiology of primary atrophic rhinitis is deserving of record. Since the term "atrophic rhinitis" is still loosely used by many physicians, it is to be emphasized that there are two clinical types of the condition:[1] (1) the primary type, known as atrophic fetid rhinitis, or ozena, and (2) the secondary type, which follows a chronic suppurative infection of the paranasal sinuses. Only primary atrophic rhinitis, or ozena, is considered in this paper.

Of the numerous theories evolved in the past, few have survived the test of time. Every one advanced has been proved and disproved. Ozena is to the rhinologist what otosclerosis is to the otologist—both conditions are chronic, both are progressive, both consist of circumscribed pathologic changes, neither gives systemic discomfort and both produce social and economic problems.

Of historical interest are the theories thus far proposed regarding the causes of ozena. Briefly summarized they are:

1. The disease is hereditary. Reichardt,[2] from a study of 27 families comprising 192 persons, of whom 27 per cent had atrophic rhinitis of the fetid type, concluded that the condition is inherited as a dominant mendelian characteristic.

2. Ozena is caused by a specific ferment.

3. It is a form of syphilis.

4. It is a chronic inflammation of the nasal mucosa with atrophy. This pathologic condition represents the secondary form of the disease.

5. It is of trophoneurotic origin (Kraupitz[3]). There is a certain relationship between the sympathetic system and ozena as a result of the disturbance of circulation caused by the disease.

6. It is nonbacterial,[4] since it is nontransmissible to other human beings.

7. A wide nose predisposes to ozena. The disease is not found in narrow noses.

8. Ozena is caused by osteomalacia.

From the Department of Laryngology, Rhinology and Otology of the University of Illinois College of Medicine, and the Research and Educational Hospital.

1. Ruskin, S. L.: Differential Diagnosis and Therapy of Atrophic Rhinitis and Ozena, Arch. Otolaryng. **15**:222 (Feb.) 1932.

2. Reichardt, H.: Monatschr. f. Ohrenh. **70**:389 (April) 1936.

3. Kraupitz, P.: Arch. f. Ohren-, Nasen- u. Kehlkofh. **138**:102 (May 17) 1934.

4. Costiniu, A., and St. Popian, L.: Oto-rhino-laryng. internat. **17**:321 (May) 1933.

9. It is due to an imbalance of cholesterol content.[5]

10. It is caused by avitaminosis.

11. It is caused by an infectious agent (Coccobacillus foetidus ozaenae [Perez[6]]). Loewenberg and Abel considered a combination of Friedländer's bacillus, pneumobacillus and Bacillus rhinoscleromatis a causative factor.

12. It is related to a hormone in the tonsils.[7]

13. It is due to an endocrine disturbance: (*a*) a dysfunction of the adrenal gland,[8] (*b*) hypoparathyroidism[9] or (*c*) hypopituitarism or dyspituitarism.

According to Wright and his co-workers,[10] cranial roentgenograms of patients with ozena gave evidence of a disturbance of function of the anterior lobe of the hypophysis, a pituitary hypofunction, indicated by sclerosis in the calvaria.

It is on this basis that the nasal osseous changes are to be understood. The anterior lobe may produce an effect on the nose, possibly through the gonads by its gonadotropic action, a vascular engorgement and swelling. The ovarian hormone exercises a specific physiologic influence on the mucous membrane of the turbinates, both during the menstrual cycle and in pregnancy. Histologically, that change is hyperemia, followed by perivascular edema in the submucosa. When this is excessive, it leads to cellular infiltration and permanent enlargement of the turbinates may result.

The present trend is toward the belief that the atrophy is due to an inherited constitutional factor, while the secondary characteristics, i. e., fetor, crusting and sinal disease, are the result of secondary infection by proteolytic organisms plus the drying effect of large drafts of air through the widened nasal passages.

We wish to cite 2 cases as additional evidence of the last quoted explanation of primary atrophic rhinitis, the first as evidence of the inherited characteristic of the disease, the second as evidence that anatomic deformity favors the development of the secondary characteristics.

The first case is that of an infant 5 months old with definite primary atrophy of the right nasal tissues and decided crusting and ozena.

F. R., a boy 5 months old, was brought to us with the complaint that for the past four months the mother had noted a foul odor from the right nostril and had removed large crusts from this area. She was much concerned, for her husband suffered from atrophic rhinitis with ozena and she believed that the child had inherited the condition.

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5. Wachsberger, A.: Laryngoscope **42**:870 (Oct.) 1932.

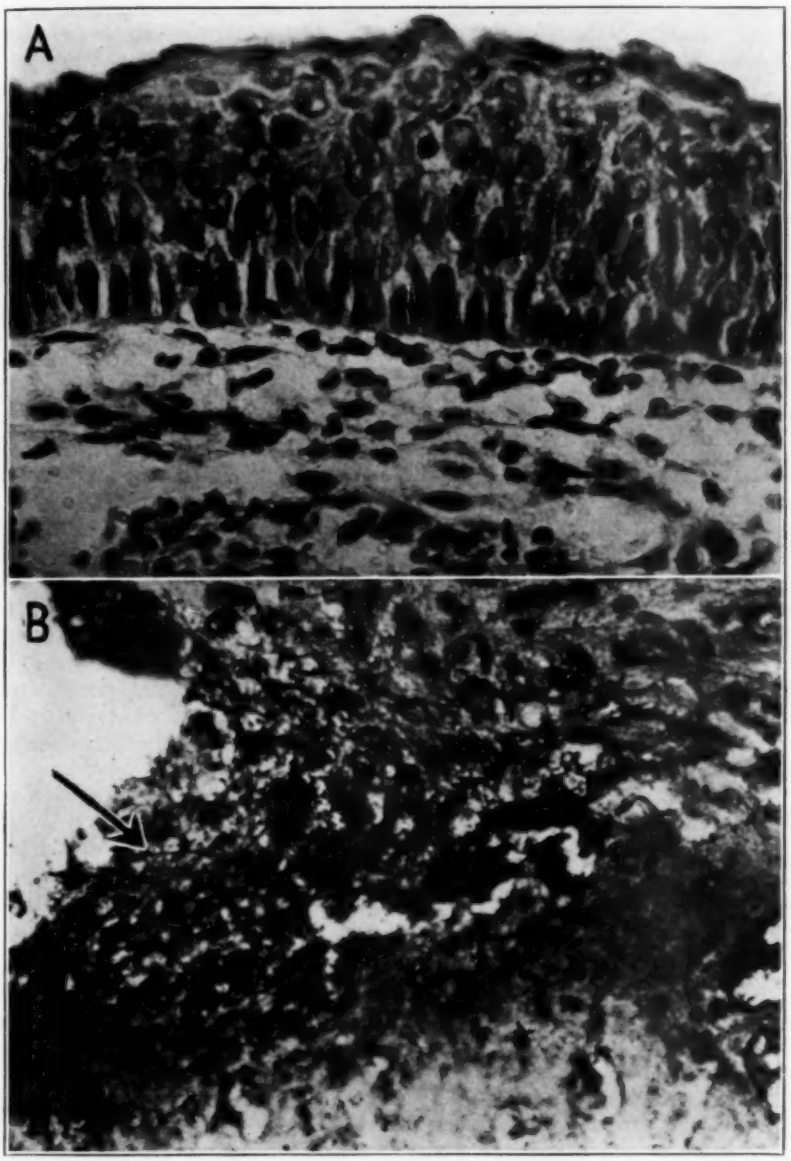
6. Perez, cited by Hofer, G.: Deutsche med. Wchnschr. **58**:1290 (Aug. 12) 1932.

7. Halász, G.: Monatschr. f. Ohrenh. **74**:347 (May) 1939.

8. Pratt, J. A.: Ann. Otol., Rhin. & Laryng. **41**:187 (March) 1932.

9. Briani, A. A.: Valsalva **10**:1 (Jan.) 1934.

10. Mortimer, H.; Wright, R. P., and Collip, J. B.: Canad. M. A. J. **37**:445 (Nov.) 1937.



A, changes in the mucous membrane of the inferior turbinate of a 5 month old child with primary atrophic rhinitis. *B*, osseous changes in the inferior turbinate of a 5 month old child with primary atrophic rhinitis.

Examination of the nose revealed extreme atrophy of the lower turbinate on the right side, which, together with the middle turbinate, was covered with crusts and pus. There was a most foul odor from this area. The left side appeared normal. Nothing in the history of the child was significant. He had been normally delivered, the first child of young parents, i. e., the mother was 22 and the father 24. The father, however, had suffered from atrophic rhinitis since early childhood. A thorough physical examination failed to reveal any disease in the parents except the father's nasal condition. The child was given a thorough examination, including Wassermann and Kahn tests of the spinal fluid and roentgenograms of the head for the detection of possible osteomyelitis, but nothing unusual was noted.

Apparently this child was born with atrophic rhinitis and ozena of the right nasal tissues. Other cases of very young children have been cited in the literature. Lermoyez [11] reported the case of a 6 month old baby who had typical purulent rhinitis. Both the mother and the father had ozena. In general, most authors believe that one is rarely able to diagnose ozena in patients under the age of 4 years.

Schmidt [12] reported 7 children in one family who had ozena. Baumgarten [13] saw 3 infants who showed tube-shaped crusts immediately after birth, but the condition could not be diagnosed as ozena until the fourth year. Birkholz [14] found 3 children between 5 and 7 years of age with ozena. The father of 1 had been treated for the condition. Raaflaub [15] stated that examinations of school children in Switzerland indicated that ozena is present in about 0.42 per cent, mostly in children who live under poor social conditions. In a comparative study, Treitel [16] found that ozena did not develop in his patients until the fifth to the sixth year, and he expressed the opinion that the condition usually does not occur after the tenth year if it develops in childhood at all. On the other hand, Mounier-Kuhn [17] maintained that among 800 patients not more than 3 or 4 under the age of 10 years could be found whose condition could be diagnosed as ozena.

That the secondary characteristics are favored by anatomic deformity is well illustrated by the alleviation of symptoms afforded by the various surgical procedures which reduce the width of the nasal passages. The surgical management of ozena with a recent modified technic has been presented by one of us (S. M. M.).[18] Additional evidence is afforded by the following case:

M. F., 28 years old, had suffered from atrophic rhinitis, with ozena, since early childhood. The discharge had been entirely from the right side, the left giving no symptoms. Examination of the nose revealed considerable atrophy of all nasal structures on the right side, with a great amount of foul-smelling crust

11. Lermoyez, cited by Flejschmann, O.: Arch. f. Ohren-, Nasen- u. Kehlkopfh. **133**:199, 1932.
12. Schmidt, V.: Monatschr. f. Ohrenh. **59**:1049, 1925.
13. Baumgarten, E.: Arch. f. Laryng. u. Rhin. **22**:492, 1909.
14. Birkholz, J.: Monatschr. f. Ohrenh. **65**:857 (July) 1931.
15. Raaflaub: Schweiz. med. Wchnschr. **57**:136 (Feb. 5) 1927.
16. Treitel, F.: Arch. f. Laryng. u. Rhin. **16**:336, 1904.
17. Mounier-Kuhn, P.: Lyon méd. **151**:672 (May 28) 1933.
18. Morwitz, S. M.: Illinois M. J. **69**:130 (Feb.) 1936.

covering all the nasal tissues. The septum was entirely subluxated to the left, causing an extreme narrowing of the left common meatus. Examination of this side revealed a fairly pale mucosa but no crusts or discharge. Roentgen examination of the sinuses disclosed a diffuse shadow throughout the right series of sinuses, while the sinuses of the left series, though not normal, were only perceptibly involved. A reposition of the septum was done, reversing the previous anatomic deformity, so that the previously widened right side was narrowed while the left side was widened. Within three months, crusts and an odor developed on the left side, which had not been involved, while on the right side the crusts and odor were definitely decreased. A roentgenogram of the sinuses showed proportionate changes.

This same procedure, i. e., a reposition of the septum, has been done by us on numerous occasions on normal persons without any resulting atrophy of the nasal tissues on the widened side.

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Clinical Notes; New Instruments and Technics

AN IMPROVED ANTRUM TROCAR

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Probably no other therapeutic procedure is as frequently and beneficially employed in the average rhinologist's office as is the irrigation of the antrum. Of all the paranasal sinuses the antrum is most often involved (according to some writers, in as many as 97 per cent of the cases of sinal disease). It is therefore worth while reemphasizing that better and more lasting results are obtained in the treatment of this sinus than in that of any of the others, either by conservative or by surgical means.[1]

In spite of the fact that the present trend is to penetrate the sinus through the natural ostium, it is nevertheless true that in many cases the older method of antral puncture is more desirable from the point of view of the physician and less irritating to the patient. Furthermore, the occasion frequently arises when the use of the puncture method is imperative. The object of this paper is to stress the advantages of antral puncture and to describe an improved antrum trocar which facilitates the procedure in several ways.

Generally speaking, there is a great divergence of opinion regarding the value of the natural ostium route. The belief is practically unanimous that this approach, for purely anatomic reasons, cannot be employed in 100 per cent of the cases, either on living patients or on cadavers. Even Merica,[2] who reported a series of 500 cases in which the maxillary sinus was irrigated through the natural or accessory ostium, admitted that there are exceptional cases in which catheterization cannot be achieved.

Van Alyea [3] showed that of 163 specimens the natural or accessory ostium was easily accessible in only 89, or 39.2 per cent. On the other hand, in 13.5 per cent cannulization through the natural ostium was impossible. Though an advocate of the use of the natural route, Van Alyea concluded that from a mechanical standpoint puncture is more efficient and that the cavity may be more thoroughly flushed out when separate openings are used for the inflow and the outflow.

Rosenberger,[4] who stated the same convictions, studied the natural ostium in 135 cadavers and in 70 consecutive private patients. Of the former he was successful in catheterizing 46 per cent; of the latter, 68 per cent. He admitted that one may be successful in catheterizing the antrum on one side and fail on the other. He enumerated the following disadvantages of cannulization through the natural ostium: (1) It cannot be universally applied; (2) there is lack of

From the Otolaryngological Service of Metropolitan Hospital, Welfare Island (Dr. J. A. W. Hetrick, director).

1. Wolf, G. D.: End Results of Rhinological Surgery, M. J. & Rec. **133**: 178-182 (Feb. 18) 1931.

2. Merica, F. W.: Irrigation of the Maxillary Sinus Through the Ostium: A Clinical Study of Five Hundred Cases, Ann. Otol., Rhin. & Laryng. **48**:1011-1119 (Dec.) 1939.

3. Van Alyea, O. E.: The Ostium Maxillare, Arch. Otolaryng. **24**:553-569 (Nov.) 1936.

4. Rosenberger, H. C.: The Clinical Availability of the Ostium Maxillare, Ann. Otol., Rhin. & Laryng. **47**:176-182 (March) 1938.

certainty that the tip of the cannula is within the antrum; (3) trauma is likely to be inflicted on the middle meatus and the ostium.

Myerson [5] presented photographs of 24 varieties of cannula for the natural orifice, to which he added one of his own, which is manufactured in three different sizes. One has the impression that the multiplicity of instruments is in itself an indication of the difficulty of cannulization. He reported that when bleeding occurred after catheterization as a result of penetration of the pars membranacea septi it lasted as long as four hours, and on rare occasions four days.

In a careful anatomic study, Simon [6] demonstrated that the so-called maxillary ostium is in the majority of cases really a canal of 3 mm. or more in length. The average length of this nasoantral canal in 110 cases was 5.55 mm. It is rather surprising, in view of these findings, that the term "natural orifice" is still employed. The fact is that a canal of significant length must be catheterized in order to penetrate the antrum through this route.

It is obvious from a review of the literature that most authorities agree that for a given percentage of patients antral puncture must be resorted to even by those most skilled in catheterization. This statement is made here chiefly for the benefit of those who are under the erroneous impression that irrigation of the antrum by puncture is an obsolete procedure. I must state unequivocally that with a proper instrument and correct technic antral puncture is painless, shockless and relatively bloodless, even for the young, the exception being those who have a phobia for instrumentation of any kind. I have frequently observed that when for technical reasons one antrum of a patient was catheterized and the other punctured he voluntarily remarked on his subsequent visit that the punctured side was more comfortable. Both methods were practiced at different times on a few patients, with their full knowledge and cooperation, and they almost invariably preferred the puncture to cannulization.

Up to 1926 I employed various antrum trocars, all of the curved type. Most of my experience has been with the Coakley instrument. In 1927 I had the opportunity to observe the use of the Lichtwitz needle and have since discarded the others. With the Lichtwitz needle or its modification I have never encountered either emphysema or infiltration of the soft tissues. I also prefer this needle for the injection of iodized oil into the antrum, because it fits the standard syringes.[7] In 1929 I modified the Lichtwitz handle.[8] In 1936 I made the further improvement of an interchangeable needle, which met with considerable approval.[9] I am offering another modification, which I have found more satisfactory than the previous ones (fig., *A*). Its chief advantage lies in the fact that instead of the beveled edge needle it has a sharp-pointed trocar. This favors a smoother, less traumatizing and easier penetration through the membranous part of the nasal wall of the antrum. This new instrument consists of two parts:

1. The handle, or grasping apparatus, consists of a shallow cup and two horizontal bars constructed to fit the curves of the fingers. A Luer-Lok is affixed to the handle. From the Luer-Lok projects a stylet, permanently attached, which

5. Myerson, M. C.: Natural Orifice of the Maxillary Sinus: Eleven Clinical Studies, Arch. Otolaryng. **15**:716-733 (May) 1932.

6. Simon, E.: Anatomy of the Opening of the Maxillary Sinus, Arch. Otolaryng. **29**:640-649 (April) 1939.

7. Wolf, G. D.: The Study of Antrum Disease with the Aid of Injection of Iodized Oil, New York State J. Med. **29**:193-198 (Feb. 15) 1929.

8. Wolf, G. D.: Modification of the Lichtwitz Antrum Trocar, Arch. Otolaryng. **9**:317 (March) 1929.

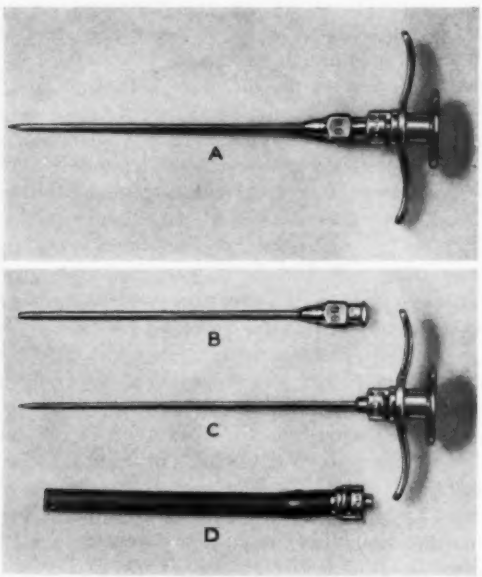
9. Wolf, G. D.: An Improved Antrum Needle, Arch. Otolaryng. **23**:229-232 (Feb.) 1936.

fits into the needle or the second section of the instrument and has a sharp point (fig., *C*).

2. The second part consists of a cannula 80 mm. long with a no. 15 gage (fig., *B*).

TECHNIC

A brief restatement of the anatomic region to be invaded is in order. The maxillary process of the inferior turbinate forms the thinnest osseous portion of the lateral wall. The membranous part is the thinnest portion of the nasal wall of the antrum. It consists of only two layers of mucous membrane, the antral and the nasal. Its anatomic boundaries are: above, the lamellae of the bulla,



A, the instrument assembled. *B*, cannula. *C*, trocar. *D*, rubber attachment.

below, the superior margin of the inferior turbinate; in front, the anterior attachment of the uncinate process, and behind, the ramus of the palate bone.

After proper anesthetization of the inferior meatus, the trocar is inserted in the following manner: It is directed close to the attachment of the inferior turbinate and by gentle pressure, without force, the puncture of the antrum through the lateral wall of the nasal cavity is accomplished with ease. By holding the cannula with the left index finger and thumb, one can remove the stylet portion of the instrument easily with the right hand by twisting the handle to the left. The syringe, irrespective of the type the operator selects, should be provided with a Luer-Lok or a similar locking device; the operator connects it with the cannula, and the irrigation of the antrum proceeds in the usual manner.

SUMMARY AND CONCLUSIONS

A new improved instrument is offered for use in puncturing the antrum.[10] Clinical experience of many years, as well as experiment on the cadaver, has

10. The instrument is manufactured by Becton, Dickinson & Co., Rutherford, N. J.

convinced me that a straight needle has numerous advantages over any curved type. I have no quarrel with those who cannulize the natural ostium. That procedure has advantages and should be used in selected cases. However, employment of a straight thin gage needle reduces the indications for cannulization. The literature on the subject is reviewed, and some of the disadvantages of catheterization are summarized.

1075 Park Avenue.

HEARING TESTS IN THE OFFICE

An Improved Technic

J. Dudley Singleton, M.D., Dallas, Texas

The difficulty of making a satisfactory hearing test in the average office, with its attendant noises, is known to almost every otologist who owns an audiometer. In order to simplify the procedure and reduce the interference from offending noises, I have devised a simple technic that helps me secure a more accurate reading in less time than was formerly required.

First, the outer portion of the external canal of the ear to be tested last is packed tightly with cotton. The patient's head is then tilted to the side, and 5 or 6 minims (0.31 to 0.37 cc.) of glycerin is dropped into the canal on the cotton, thus making a very effective plug. More cotton may be used to absorb any excess of glycerin. Next, the patient is shown how to hold the receiver over the ear which is to be tested. He is then told that he will hear a low pitch sound, faintly at first but in rapidly increasing volume, so that he will have no difficulty in hearing and identifying the sound.

In beginning the test, the volume is set well above the patient's auditory threshold and decreased 5 decibels at a time. The current interrupter is used to prevent nerve fatigue and the carry-over of sound. When the patient fails to hear, the volume is increased to the point where sound was last heard and then decreased by 2 or 3 decibels until the patient's auditory threshold is definitely established. In order to avoid errors, the patient should be required to identify the sound at the threshold point three or four times. With each change of frequency he is told that he will hear a different sound, and the procedure is followed until the test is completed. The other ear is tested in the same manner.

COMMENT

The advantages of the technic described are as follows:

1. The cotton and glycerin plug is simple, easy to use and fairly effective in reducing and eliminating offending noises.

2. The strong volume used at the beginning of each frequency, along with the current interrupter, familiarizes the patient with the sound and prevents fatigue and carry-over.

3. The patient recognizes his auditory threshold more quickly and accurately when it is approached from above.

Medical Arts Building.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

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PERORAL ENDOSCOPY

LOUIS H. CLERF, M.D.
PHILADELPHIA

ANESTHESIA

Three oustanding requirements in peroral endoscopic procedures are of extreme importance: safety, efficiency and comfort. Adequate anesthesia will contribute substantially to the fulfilment of these. Jackson and McReynolds [1] in a survey of the experiences and opinions of peroral endoscopists found considerable difference of opinion regarding what constitutes the most desirable plan of anesthesia for these procedures. Morphine, without or with atropine, long regarded as the standard agent of premedication, still holds first place. Next to morphine in popularity are barbiturates. A few use scopolamine with morphine. Local anesthesia still is employed by the majority in preference to general anesthesia. For direct laryngoscopic procedures the anesthetic is either applied with a swab to the piriform sinuses or administered in some form of spray or by instillation or both. A few use a brush for application.

For bronchoscopic work the majority use the same technic in the induction of anesthesia as for direct laryngoscopic procedures, combining this technic with intratracheal instillations. For esophagoscopy about one half of those who responded to a questionnaire used the same technic as for direct laryngoscopic and bronchoscopic procedures. Several employed an anesthetic solution as a gargle, and one used avertin with amylene hydrate rectally. Five endoscopists employed no anesthetic in esophagoscopy.

Various opinions on the advantages and disadvantages of the different drugs used for the induction of local anesthesia and opinions on certain of the barbiturates have been tabulated and summarized in the article. With regard to the anesthetizing agents employed, over 50 per cent of the endoscopists still prefer cocaine; 8 indicated that this drug is not used at all. Other drugs employed, in their order of frequency, are pontocaine, larocaine, butyn, metycaine, metrabol, ethyl aminobenzoate

1. Jackson, C. L., and McReynolds, G. S.: Anesthesia for Peroral Endoscopy, Ann. Otol., Rhin. & Laryng. **41**:1048 (Dec.) 1940.

and Forestier's solution.[1a] Avertin with amylene hydrate was approved by 16, condemned by 14 and not used by 17. Other barbiturates are used with less frequency and also are not used by the greater number. General anesthesia is employed by some for children. It is of interest that a number expressed the belief that general anesthesia is more necessary for esophagoscopy than for other procedures. Three use general anesthesia for all esophagoscopic procedures, while 9 use it only in the presence of sharp foreign bodies in the esophagus.

The authors set forth their technic with regard to anesthesia. They employ morphine and atropine, supplemented at times by pentobarbital sodium, preliminary to the majority of their endoscopic procedures. For patients undergoing a series of endoscopic procedures, particularly if they are treated as outpatients, premedication is dispensed with. For direct laryngoscopic and bronchoscopic work the pharynx is sprayed with a 10 per cent solution of either cocaine or larocaine hydrochloride. Pontocaine is occasionally used, but its use is contraindicated for asthmatic patients. After pharyngeal anesthesia has been established, fractional instillations of one of the previously noted solutions are made into the larynx or trachea by the aid of a mirror laryngoscope. For supplemental anesthesia, an intrabronchial spray or application of a concentrated solution through the bronchoscope is used. Swab applications to the piriform sinuses have been practically discontinued. For esophagoscopy in adults, a simple spray to the pharynx usually suffices. For children under 8 years of age, usually no anesthetic agent, local or general, is employed, although because of its apparent nontoxic effect larocaine may be used.

In summarizing his experiences with anesthesia in peroral endoscopy, Nail [2] expressed the opinion that practically all operations for foreign body and even some for pulmonary disease in children under 12 years of age are better carried out with the patient under anesthesia induced by avertin with amylene hydrate than with the region under local anesthesia. Avertin with amylene hydrate used according to the 80 mg. table, alone or in conjunction with larocaine hydrochloride, produces safe and adequate anesthesia for difficult extractions of foreign bodies in children or adults. For routine bronchoscopic procedures in adults, he supple-

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1a. Forestier's solution is made up as follows:

	Gm. or Cc.
Cocaine hydrochloride	2.00
Phenol	.20
Solution of epinephrine hydrochloride (1 : 1,000)	10.00
Cherry laurel water	40.00
Distilled water to make	150.00

2. Nail, J. B.: Anesthesia in Peroral Endoscopy, Texas State J. Med. **36**:430 (Oct.) 1940.

mented the topical application to the piriform sinuses and the intratracheal instillation of larocaine hydrochloride with instillation through a catheter inserted into the trachea of 1 cc. of a 2.5 per cent solution of larocaine hydrochloride into each main bronchus. With this method, additional instillations through the bronchoscope are not required.

CONGENITAL ANOMALY

Dyspnea.—Schall and Johnson [3] reported an interesting case of respiratory obstruction due to a congenital anomaly of the aorta in a child 6 months of age. The patient had been well until a respiratory infection developed two weeks before admission to the hospital. Noisy wheezing respiration, slight cough and almost complete loss of voice were observed. On several occasions there were attacks of cyanosis. Physical examination gave practically negative results except for the previously noted findings. Roentgen study of the chest revealed essentially normal conditions. By direct laryngoscopic examination it was observed that laryngeal motility was normal, that there was no evidence of obstruction of the larynx and that there was slight redness of the posterior surface of the arytenoids. The child was seen again sixteen days later. At that time there had been no change in the symptoms. The larynx exhibited no abnormality. Bronchoscopic examination revealed narrowing of the trachea immediately above the carina. This appeared to be due to a swelling of the mucous membrane. There was no abnormal pulsation. When the bronchoscope was passed beyond the point of narrowing, respirations were quiet, but after removal of the bronchoscope the patient again became cyanotic, and respirations became difficult. Tracheotomy was performed with relatively little improvement in the breathing. It was noted on repeated bronchoscopic examinations that as soon as the bronchoscope was passed beyond the point of narrowing, all the symptoms cleared up. A cane-shaped cannula was substituted, with relief of the symptoms. Ten days later the patient had a massive pulmonary hemorrhage and died.

At autopsy there was found a persistence of the embryonic fourth arches, forming a left and a right aortic arch, which together formed a circle enclosing within it the trachea and esophagus and producing the previously described obstruction of the trachea above the carina. The left carotid and subclavian arteries arose in the usual manner from the left aortic arch. The innominate artery was absent, and in its place the right carotid and subclavian arteries arose as distinct vessels from the right aortic arch.

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3. Schall, L. A., and Johnson, L. G.: Dyspnea Due to Congenital Anomaly of the Aorta, Ann. Otol., Rhin. & Laryng. **41**:1055 (Dec.) 1940.

The authors discussed the embryologic anatomy and the comparative development of the aortic arches and noted that in an analysis of 1,000 congenital cardiac anomalies 5 similar cases had been reported. The diagnosis of this condition during life is difficult. However, peculiar wheezing, cyanosis on exertion and a staccato cough in the absence of any other cause suggest the possibility of some anomaly of the aortic arch.

Hemangioma of the Larynx.—Although hemangioma of the larynx is not of common occurrence, the infantile type, which usually occurs subglottically, is rare. In spite of this, however, it should be considered as a diagnostic possibility, as shown in the case reported by Suehs and Herbut.[4] An infant aged 14 weeks had been well until three weeks previously, when slight hoarseness and noisy breathing were observed. There was no fever. These symptoms continued unchanged for about two weeks, when periods of marked respiratory difficulty occurred, which lasted for several hours and then cleared up. These recurred with increasing severity as well as frequency, and finally the child was admitted to the hospital in the midst of a severe attack of dyspnea, which necessitated immediate tracheotomy. Although the dyspnea was relieved, death resulted within twenty-four hours.

At autopsy there was found below the left vocal chord a fusiform submucosal tumor, which almost completely obliterated the laryngeal airway. The tumor consisted of closely packed mature and immature capillaries, conforming to a simple cellular hemangioma.

ACUTE LARYNGOTRACHEOBRONCHITIS

If all cases of laryngeal dyspnea were considered as cases of laryngotracheobronchitis, the number demonstrated to be the latter would be increased tremendously. Brighton [5] preferred to consider as cases of acute laryngotracheobronchitis only those cases in which the obstructive laryngeal dyspnea was sufficient to warrant tracheotomy. In reviewing the literature he found that recent mortality statistics are still practically the same as those noted in the earlier reports. Treatment is difficult, because the diagnosis depends on the condition of the larynx and the tracheal mucosa after the disease has become established.

Brighton outlined the treatment employed as a routine procedure in cases of obstructive laryngeal dyspnea. Humidification of the oxygen tent and recently the use of helium have aided materially in postponing and at times avoiding the necessity for tracheotomy. During the past

4. Suehs, O. W., and Herbut, P. A.: Hemangioma of the Larynx in Infants, Arch. Otolaryng. **32**:783 (Oct.) 1940.

5. Brighton, G. R.: Laryngotracheobronchitis, Ann. Otol., Rhin. & Laryng. **41**:1070 (Dec.) 1940.

three years chemotherapy has been employed routinely. If tracheotomy is done, secretions are aspirated through the cannula. He employed a 1 per cent solution of ephedrine in physiologic solution of sodium chloride for instillation and recently has used a 5 per cent sterile solution of sodium bicarbonate prior to the aspiration of secretions with a mechanical aspirator.

Reference is made to some of the findings in cases of laryngotracheobronchitis in fowls. A remarkable similarity has been observed in the results of histologic studies carried out on chickens and on children. Sections representative of both groups showed marked infiltration with mononuclear cells, a finding which is characteristic of the tissue response to virus disease. While no vaccine or serum has been used on the human beings, it would seem feasible to concentrate on the problem of finding suitable preventive agents rather than simply to employ the mechanical aids now in use. While chemotherapy appears to have little effect on the mortality in acute stages of the disease, Brighton stated the belief that it should be continued and should be supplemented by the use of vaccines or of transfusions of blood from patients who have recovered from acute laryngotracheobronchitis.

In discussing the treatment of acute laryngotracheobronchitis, Davison [6] expressed agreement with other writers regarding the importance of humidification of the air. To avoid the resulting high temperature when a steam-humidified croup tent was used, he employed an electrically operated humidifier and modified it so that it could be used to moisten the air in the canopy of an ordinary oxygen tent. By this means it was possible to maintain a relative humidity of 95 per cent, while the temperature of the tent remained at 68 or 70 F. A series of 17 cases was reported, with a mortality rate of 11.7 per cent.

He concluded that subglottic edema is less marked if the patient is kept in cool moist air rather than in warm moist air. The development of pneumonia usually can be prevented if treatment is instituted early and if the bronchi are kept open at all times by removal of obstructing secretions and crusts. Atropine and sedatives not only are unnecessary but are definitely contraindicated. Direct laryngoscopic examination is necessary for accurate diagnosis, and bronchoscopic equipment is essential in the management of patients with this disease. Sulfanilamide is of value when Streptococcus haemolyticus is the infecting agent.

After a further study, to determine the ideal climatic conditions for the treatment of patients with laryngotracheobronchitis, Davison [7] con-

6. Davison, F. W.: Treatment of Acute Laryngotracheobronchitis, Arch. Otolaryng. **32**:321 (Aug.) 1940.

7. Davison, F. W.: Some Observations on the Control of Temperature and Humidity in Oxygen Tents, Ann. Otol., Rhin. & Laryng. **49**:1083 (Dec.) 1940.

cluded that the temperature and humidity of an oxygen tent exert a greater effect on the comfort of the average patient than does the concentration of oxygen. For this reason a wet and dry bulb thermometer should be part of the equipment of every oxygen tent. By the employment of a mechanical humidifier, the humidity in the tent can be raised to 95 per cent when the temperature is 70 F. Temperatures from 68 to 70 F. are most comfortable when the humidity is high. An ice-cooled oxygen tent equipped with a motor is a useful and practical means of increasing loss of heat from the body. Loss of water from the body can be decreased by increasing the humidity if the temperature of the air is not above 70 F.

BRONCHOSCOPY FOR DIAGNOSIS

Bronchial Obstruction.—In a general discussion of the value of bronchoscopy in diagnosis, Hart [8] emphasized the importance of bronchoscopic examination in the study of patients exhibiting evidences of obstructive emphysema or obstructive atelectasis. In any case of bronchial obstruction in which the diagnosis cannot be made promptly by the ordinary methods, including roentgen examination, bronchoscopy should be resorted to without delay. A number of illustrative cases are cited.

The factors most commonly found as causes of obstructive emphysema are foreign body, tumor and accumulated secretion. In a discussion of this subject Maxwell [9] cited 6 illustrative cases. These were divided into two groups. In the first the emphysema was transient, resolved spontaneously and usually was due to thick secretion. In the second group the obstruction was permanent, the common cause being tumor. The emphysema was nearly always temporary and was followed by atelectasis. Diagnosis of emphysema by physical examination alone may be difficult, but if the physical examination is supplemented by proper roentgen studies, the condition should be recognized readily. Since emphysema is an early sign of bronchial obstruction, the finding of the condition should lead at once to a consideration of bronchoscopy and if necessary the instillation of iodized oil.

The bronchoscopic observation of collapse of a main bronchus without evidence of external pressure is uncommon. In a search of the literature Bozer [10] was unable to find any reference to this subject. He

8. Hart, V. K.: Importance of Bronchoscopy in the Diagnosis of Intrathoracic Conditions, North Carolina M. J. **1**:331 (July) 1940.

9. Maxwell, J.: Localized Emphysema as a Sign of Incomplete Bronchial Obstruction, Brit. M. J. **1**:520 (March 30) 1940.

10. Bozer, H. E.: Bronchial Collapse Without Clinical Signs of External Pressure: Bronchoscopic Observations, Ann. Otol., Rhin. & Laryng. **49**:1091 (Dec.) 1940.

reported 3 cases in which there was clinical and roentgen evidence of atelectasis of one lung. At bronchoscopic examination there was found collapse of the bronchus without any evidence of endobronchial changes which would account for the obstruction. The bronchial walls appeared flaccid, and with the aid of the bronchoscope and an expanding forceps it was possible to open the bronchus. In 2 of the cases death resulted. Unfortunately, permission was not secured for an autopsy. Therefore, no conclusions could be arrived at. In the case of the third patient there was a history of allergy, and it is possible that the collapse of the bronchus was due to bronchospasm. In this case obstruction was not complete, and at a second bronchoscopy, performed one week later, the bronchial tree appeared normal. Bozer assumed that two mechanisms might be considered as responsible in this case—(1) an exceedingly strong spasm of the bronchial musculature and (2) a structural weakness of the bronchial wall.

A localized stricture-like narrowing of a bronchus as a complication of either allergic or infectious asthma is more common than is generally believed. In a group of 140 patients suffering from asthma Prickman and Moersch [11] observed and treated 60 who were found to have definite stenosis of one or more bronchi. A study of these revealed that bronchostenosis usually produces characteristic symptoms as well as physical and roentgenologic changes.

Severe, persistent, often paroxysmal cough is one of the prominent symptoms. Frequently it is difficult for the patient to raise sputum until after prolonged coughing. Fever of moderate severity, with or without chills, occurred in 69 per cent of these patients. It is of interest that 53 per cent of those with bronchostenosis gave a history of previous pneumonia. The physical signs and roentgen findings were those commonly observed in atelectasis. These usually were confined to the lower and posterior portions of the chest. The bronchoscopic evidence was a narrowing of one or more bronchi, due apparently to an inflammatory process and unrelated to allergic edema or bronchial spasm. It was common to see thick tenacious mucus and at times purulent secretions, which contributed to the bronchial narrowing and often were retained in considerable quantity beyond the point of narrowing.

Treatment consisted of dilation of the stenotic bronchus by means of a forceps introduced bronchoscopically and aspiration of retained secretions. The instillation of medicaments did not appear beneficial and was discontinued. It is obvious that the recognition of this condition and the employment of appropriate treatment are dependent on close cooperation between the internist and the bronchoscopist.

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11. Prickman, L. E., and Moersch, H. J.: Bronchostenosis Complicating Allergic and Infectious Asthma, Ann. Int. Med. **14**:387 (Sept.) 1940.

Diffuse inflammatory tracheobronchial obstruction is of rare occurrence. In the case reported by Samson,[12] that of a man aged 71 years, the symptoms were of eight years' duration and were progressive. They consisted of paroxysmal cough, which later was almost constant, raising of purulent sputum in increasing quantity, progressive hoarseness and loss of weight. The physical signs were not remarkable. The roentgen findings suggested a chronic infection in the upper part of the right lung, with fibrosis. At bronchoscopic examination the tracheal wall was found to be covered with discrete and confluent masses of reddish yellow polypoid tissue, which were slightly elevated above the surface. These changes appeared more extensive at the lower end of the trachea and continued into both bronchi, particularly the right. Numerous specimens of tissue were scalped off with the tip of the bronchoscope. After this there was improvement in cough. The patient left the hospital and subsequently died. At autopsy the diffuse polypoid changes previously observed on bronchoscopic inspection were found to extend into the smaller bronchi.

In the treatment of laryngeal obstruction, relief of asphyxia is the most important problem. In discussing this, Galloway [13] pointed out that long-continued obstruction leads to marked changes, which are secondary to continuously increased negative intrathoracic pressure. These consist of congestion, edema, hemorrhage, atelectasis and emphysema. Superimposed infection may give rise to atypical pneumonia, pneumonitis, pulmonary abscess or bronchiectasis. Therefore intervention must not be delayed until these irremediable changes are established.

TUMORS OF THE TRACHEA AND BRONCHI

Carcinoma of the Trachea.—The occurrence of carcinoma of the trachea in a man aged 65 years was reported by Miller.[14] The initial symptoms were a tickling sensation in the throat and hoarseness. Paralysis of the right vocal cord was found. A provisional diagnosis of carcinoma was made by laryngoscopic and roentgenologic examinations and was confirmed by bronchoscopic inspection and biopsy. As a rule, cancer of the trachea is not diagnosed until there are symptoms of obstruction or paralysis of the larynx with vocal disturbances. The importance of bronchoscopic examination when symptoms and clinical findings are obscure was emphasized.

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12. Samson, P. C.: Diffuse Polypoid Laryngotracheobronchitis, with a Preliminary Note on Endoscopic Curettage, J. Thoracic Surg. **9**:679 (Aug.) 1940.

13. Galloway, T. C.: Laryngotracheobronchial Obstructions and Their Secondary Effects, Ohio State M. J. **36**:851 (Aug.) 1940.

14. Miller, I.: Primary Carcinoma of the Trachea, Arch. Otolaryng. **31**:675 (April) 1940.

Adenoma of the Bronchus.—In a report of their experience with the different clinical, pathologic and therapeutic features of adenoma and malignant polypoid bronchial tumors, Brunn and Goldman [15] compared the observed features of the two types of tumors arising from bronchial epithelium—namely, adenoma and carcinoma. Adenoma is more common among females and usually is observed before the age of 40. The greatest incidence of carcinoma occurs from 40 to 60 years, and the condition is more often observed in males. In discussing the diagnostic procedures, they stated that cancer and adenoma in the early stages may give no demonstrable roentgen findings. Tomography may aid greatly in diagnosis, particularly in visualizing those tumors that cannot be seen by bronchoscopy and probably also in ascertaining whether the tumor is entirely endobronchial or has extended beyond the bronchial lumen.

The bronchoscopic image of an adenoma is often sufficiently different from that of a cancer in the same location that the lesions may be distinguished. A movable soft fleshy vascular pink or purplish polypoid mass is characteristic of adenoma. A fixed, pale yellow or gray, ulcerated, less vascular, broadly attached protruding tumor, which is indurated even to the hardness of cartilage, is the usual form of endobronchial cancer. When adenoma is present, the carina is sharp, although tilted to the side of the tumor. Cancer usually produces a flattening of the carina, and the characteristic "feel" of a "frozen" condition of the mediastinal structures is imparted to the bronchoscopist by the large extrabronchial infiltrating cancerous mass. When a biopsy specimen is taken from adenoma, bleeding is copious and frequently alarming; removal of a similar specimen from cancer produces no unexpected amount of hemorrhage. After a specimen for biopsy has been taken from adenoma, bronchoscopic observations show healing of the wound; after the same procedure on a polypoid cancer no healing takes place but the tumor enlarges and the wound fills with exuberant new growth. After local bronchoscopic removal of adenoma, a smooth mucous membrane regenerates over the former bronchial attachment, and the lumen may remain patent for years. After a similar procedure for polypoid cancer, new tumor, obstructing the bronchial lumen, is formed again in about one or two months.

Bronchoscopic biopsy may be misleading and inconclusive, because characteristic patterns often are not obtained. A superficial specimen may show only squamous cells of the overlying metaplastic mucous membrane, and so be called erroneously epidermoid cancer when an adenoma hides below. Often a crushed specimen of the epithelial cells gives the appearance of a wildly growing small cell cancer. Similarly,

15. Brunn, H., and Goldman, A.: The Differentiation of Benign from Malignant Polypoid Bronchial Tumors, Surg., Gynec. & Obst. **71**:703 (Dec.) 1940.

biopsy of a small cell carcinoma may be confused easily with adenoma. A bronchoscopic biopsy, therefore, should not be the sole foundation of judgment or the only criterion of future therapy.

Adenoma responds readily to bronchoscopic fulguration, piecemeal removal and removal by snare. Intramural or endo-extrabronchial forms cannot be removed in toto by any bronchoscopic method. In the opinion of Brunn and Goldman, bronchoscopic removal is indicated chiefly as a preliminary treatment, to reestablish bronchial drainage. The case histories of several patients are given in detail.

Laff [16] added 3 cases to the literature and reported a fourth case of adenoma of the bronchus, in which bronchial stenosis developed after treatment. He expressed agreement with other bronchoscopists regarding the importance of bronchoscopic examination in diagnosis. While bronchography is of value in certain cases of this type it gives no information concerning the actual cause of the obstruction. In view of the tendency to hemorrhage, artificial pneumothorax should be considered prior to bronchoscopic removal or destruction by electrocoagulation. Treatment by bronchoscopic means has resulted in many apparent cures or arrests and therefore should be given a trial before resort is taken in lobectomy or pneumonectomy.

In a discussion of bronchial adenoma, Goldman [17] emphasized the importance of the reestablishment of bronchial drainage in treatment. This usually can be accomplished by bronchoscopic removal of the endobronchial portion of the tumor. If disabling symptoms persist, on account of previous pulmonary suppuration, lobectomy or pneumonectomy may be required. In his experience it did not appear necessary for the maintenance of life to remove the tumor completely. Residual mural portions of the tumor apparently remained quiescent for years. Of 18 patients, 13 still are living. Of these, 2 had pneumonectomy and 1 lobectomy. In 4 the tumor was removed bronchoscopically, and 6 received radiation therapy. There were 5 deaths, 1 after pneumonectomy, 1 after bronchoscopic cauterization and 1 after many bronchoscopic drainages. In the last case the tumor had been removed. The remaining 2 patients received no treatment, and the diagnosis was made at necropsy. An excellent bibliography is appended to the article.

Carcinoma of the Bronchus.—An exact diagnosis of bronchogenic carcinoma cannot be established in every instance. Often the clinical picture suggests either an inflammatory lesion or a carcinoma, and many instances are recorded in which the clinical findings of suppuration predominated and the underlying tumor responsible for the suppurative

16. Laff, H. I.: Benign Tumors of Bronchi, with Special Reference to Vascular Adenoma, Arch. Otolaryng. **31**:148 (Jan.) 1940.

17. Goldman, A.: Polypoid Bronchial Tumors, with Special Reference to Bronchial Adenomas, California & West. Med. **52**:123 (Sept.) 1940.

lesions was overlooked. Freedlander and Wolpaw [18] presented their experiences with a group of patients in whom the opposite conditions prevailed, an inflammatory lesion being mistaken for and treated as bronchogenic carcinoma. Detailed reports of 4 cases are recorded. In 3 the clinical diagnosis of carcinoma could not be corroborated by bronchoscopic examination and other studies. In the fourth case the clinical and roentgen evidence was supported by the pathologist's diagnosis of carcinoma on the material removed by bronchoscope. It is of interest that in all of these cases the operative diagnosis was carcinoma and the diseased lung tissue was extirpated. The inflammatory nature of the lesions was recognized only after the pathologist studied the tissue removed surgically. This is an important article and merits study by all who are interested in the diagnosis and treatment of bronchial cancer as well as other pulmonary diseases.

Primary carcinoma of the bronchus now comprises approximately 10 per cent of all neoplasms diagnosed as carcinoma. Since the best results are secured by surgical removal, it is important that the diagnosis be made early if a cure is to be expected. In discussing the importance of early diagnosis, Samson and Holman [19] stated that bronchoscopic examination with biopsy probably is the most important single procedure both for diagnosis and for determining operability. This examination always should be advised without hesitancy, for in competent hands the hazards and discomforts are minimal.

While the procedure is not infallible, since visual impressions may not be substantiated by histologic examination of tissue removed, repeated bronchoscopic examinations can be made and additional tissue secured if the signs and symptoms warrant a provisional diagnosis of cancer. In approximately 75 per cent of cases a definite diagnosis can be established bronchoscopically. Bronchoscopic examination also gives valuable aid in judging the possibility of carinal fixation. The location of the tumor, the presence or absence of fixation of a bronchus or carina and the presence of submucosal extension proximal to the tumor are important factors in making a decision.

In summarizing the experiences gained in a study of 104 patients with primary carcinoma of the lung, Overholt [20] discussed the symptoms, diagnostic procedures, evidences of inoperability and end results. With

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18. Freedlander, S. I., and Wolpaw, S. E.: Chronic Inflammatory Lesions of the Lung Simulating Bronchogenic Carcinoma, J. Thoracic Surg. **9**:530 (June) 1940.

19. Samson, P. C., and Holman, E.: Primary Carcinoma of the Bronchus: The Importance of Early Diagnosis in Increased Operability and Curability, California & West. Med. **53**:208 (Nov.) 1940.

20. Overholt, R. H.: Curability of Primary Carcinoma of the Lung: Early Recognition and Management, Surg., Gynec. & Obst. **70**:479 (Feb., no. 2A) 1940.

regard to bronchoscopy as an aid in diagnosis he stated that this procedure has yielded tissue for biopsy in 72 per cent of all cases in which examination was made by this method. Since the majority of lesions are found within the range of bronchoscopic visibility, this procedure becomes the most important diagnostic method available. Two other facts make bronchoscopy invaluable. 1. A diagnosis can be made earlier by this method than by any other, for a lesion too small to occlude the bronchus and produce roentgenographic changes may be seen bronchoscopically. 2. This procedure is by far the safest method of securing a specimen for biopsy from a lesion of the bronchial stem.

Overholt concluded:

1. The medical profession should become interested in primary carcinoma of the lung, first because it is a common disease, and second, because curative treatment is available to those whose disease is still local in its extent.

2. An optimistic attitude as to the future seems justified for the following reasons: (*a*) Early warning symptoms do occur in the majority of cases; (*b*) early lesions may be suspected after roentgenographic examination, as the lung fields lend themselves well to contrasting shadows either of the tumor or of atelectasis; (*c*) three fourths of all primary lung tumors are situated in the stem bronchi, and are therefore accessible to bronchoscopic visualization and biopsy.

3. In a period shorter than a decade of surgical treatment, operability has surpassed that of the esophagus and possibly the stomach. In a series of 104 cases, 21 per cent of the lesions were considered resectable and surgical treatment was carried out. The theoretical cure rate in this series of patients has been computed to be 14 per cent. Eleven per cent of the verified cases have lived following surgical resection for varying periods of time up to 5 years and 10 months without evidence of recurrence.

Bronchoscopic diagnosis is dependent on the interpretation of pathologic conditions revealed by direct inspection of the tracheobronchial tree. The use of the bronchoscope in the diagnosis of bronchial carcinoma, as stated by Holinger and Radner,[21] is fourfold:

1. To study the character of the lesion.
2. To note the location and extent along the bronchial wall.
3. To secure tissue for biopsy if the tumor can be visualized.
4. To aid in determining the operability of the tumor.

Bronchoscopy is an indispensable aid in the differential diagnosis of certain bronchopulmonary diseases. While several recoveries follow the endoscopic treatment of carcinoma have been reported, this method is for the most part palliative.

In a study of 128 deaths due to cancer, Mather and Stoderman [22] found that bronchial cancer ranked second, contributing 18.7 per cent of

21. Holinger, P., and Radner, D. B.: Bronchoscopic Diagnosis of Bronchial Carcinoma, Surgery **8**:939 (Dec.) 1940.

22. Mather, P. P., and Stoderman, A. H.: Bronchogenic Carcinoma: A Review of Thirty Verified Cases, U. S. Nav. M. Bull. **38**:541 (Oct.) 1940.

the deaths. In the early stages neither the symptoms nor the clinical findings give a characteristic picture, and since in many of the cases the cancer occurs in the hilar region, roentgen findings also may give little information in the early stages of the disease. Early bronchoscopic examination is considered invaluable and in the absence of positive findings should be repeated. Positive bronchoscopic findings, with biopsies, were reported in 60.7 per cent of their cases.

With the more extensive use of bronchoscopy and biopsy, there has been an increase in the reported incidence of primary bronchial carcinoma. In a study of 65 cases of bronchial tumor, Broyles and Fisher [23] observed that bronchial cancer occurred most frequently between the ages of 50 and 60 years. Males were affected more often than females, in the ratio of 5:1. In their opinion, bronchoscopic examination and roentgen studies afforded the greatest aids in diagnosis, and surgical removal appeared to offer the best therapeutic results.

Of a series of 20 patients with carcinoma of the bronchus observed by Harrill,[24] biopsy specimens showing carcinoma were obtained by bronchoscopy from 17. Not only was this procedure found important as a diagnostic aid, but it furnished additional information to the thoracic surgeon regarding the endobronchial location of the tumor and the selection of those best suited for operation. In this group but 1 tumor was considered suitable for surgical extirpation. While there has been a great increase in the number of cancers diagnosed during life, the number suitable for surgical treatment is relatively small.

Bronchiogenic carcinoma is known to occur during early life. In the case reported by Wasch and others,[25] a diagnosis of adenocarcinoma involving the bronchus of an upper lobe had been made by bronchoscopic biopsy in 1932, when the patient, a boy, was 11 years of age. He was given roentgen, as well as radium, therapy and made fairly satisfactory progress until 1935, when it was again necessary to give him a course of radiation treatment. After this the patient felt well and gained considerably in weight and stature until 1939. During this interval it was noticed that his growth was exceptionally rapid and that there was marked asymmetry of the chest. In 1939 death resulted from cardiac failure. At necropsy there was found extensive visceral metastasis, the metastatic carcinoma in the heart and viscera being similar to that observed in the original biopsy specimen. There was a coincidental adenoma, associated with gigantism.

23. Broyles, E. N., and Fisher, G. E.: Bronchoscopic Experiences with Lung Tumors, Surgery **7**:918 (June) 1940.

24. Harrill, J. A.: Carcinoma of the Bronchus: Some Findings in Twenty Cases, North Carolina M. J. **1**:180 (April) 1940.

25. Wasch, M. G.; Lederer, M., and Epstein, B. S.: Bronchiogenic Carcinoma of Seven Years' Duration in an Eleven Year Old Boy, J. Pediat. **17**:521 (Oct.) 1940.

In presenting data bearing on the diagnosis of cancer of the bronchus, Hammond [26] stated that in his series of 40 cases an average of six months elapsed between the first complaint and the establishment of the diagnosis. Cough was the most common first symptom noted. Bronchoscopy was employed at one time or another in 39 of the 40 cases. An endobronchial tumor was visualized in 34 cases, in all of which biopsy was done, and a positive histologic diagnosis of carcinoma was made in 33. Of the 5 cases in which no endobronchial tumor was observed, fixation and distortion of the tracheobronchial tree were noted in 4.

Of the 26 patients treated by some form of radiation, 15 by a combination of radium, applied intrabronchially, and roentgen rays and 11 by roentgen rays alone, 22 died, the longest survival being for thirty-two months. The 4 patients who survived were treated by radium, intrabronchially applied, and roentgen rays. The radium in a suitable tube was introduced bronchoscopically and placed in the bronchus directly opposite the lesion.

In the treatment of primary bronchial carcinoma it is necessary to know not only the type but also the location and extension of the lesion into the bronchial wall. By serial bronchography under roentgenographic control, Fariñas [27] was able to obtain this information, which cannot be secured by any other method. Recently he has employed solutions of organic salts, such as iopax, as a substitute for iodized oil. The disadvantages of iodized oil, namely, its long retention in the parenchyma of the lung and its inability to enter through narrowed bronchial orifices, are overcome by the use of organic salts of iodine. These are not toxic or irritating, are rapidly absorbed by the bronchoalveolar mucosa and are passed readily through even the narrowest of stenotic bronchi. Since such salts may be passed readily through bronchial lumens, it is necessary to take the bronchograms while they are being injected. The article is illustrated with excellent bronchograms and detailed case histories.

POSTOPERATIVE PULMONARY ATELECTASIS

In a discussion of the many details which must be attended to before and during anesthesia, as well as after operation, to reduce the incidence of postoperative atelectasis, Mousel [28] stressed the importance of aspiration if the patient has an excessive quantity of secretions in the airway at the termination of anesthesia or if he has vomited during anesthesia.

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26. Hammond, A. E.: Diagnosis and Treatment of Carcinoma of the Lung, with Statistical Review of Forty Cases, Arch. Otolaryng. **31**:780 (May) 1940.

27. Fariñas, P. L.: Recent Progress in the Bronchographic Examination of Bronchogenic Carcinoma, Am. J. Roentgenol. **44**:370 (Sept.) 1940.

28. Mousel, L. H.: Postoperative Atelectasis: The Anesthetist's Part in the Diagnosis and Treatment, J. A. M. A. **115**:899 (Sept. 14) 1940.

This is best carried out bronchoscopically, so that the bronchus of each separate lobe can be visualized and cleansed of secretions. If atelectasis occurs postoperatively, the obstructing secretions should be removed as soon as the condition is discovered, for if atelectasis is allowed to persist for many hours, secondary pneumonitis or pulmonary suppuration is likely to develop. After bronchoscopic aspiration of secretions from each visible bronchial orifice, the patient should be turned frequently from side to side and cough should be encouraged.

While no one factor is believed responsible for postoperative pulmonary complications, an analysis of etiologic factors indicated to Gordon [29] that the bronchial obstruction produced by an accumulation of mucus and the effects of respiratory infection are the most potent factors. Atelectasis resulting from obstruction of a bronchus by mucus is the basis of most of these complications. Pneumonia and pulmonary abscess represent advanced stages of the primary atelectatic process. While the diagnosis should be relatively easy, the clinical findings are variable, roentgenograms often are misinterpreted and an erroneous diagnosis of pneumonia is often made when only atelectasis is present. The time of onset is of importance in a differential diagnosis.

Bronchial drainage, with aeration of the lung, is essential if atelectasis and its more serious sequelae are to be prevented. This can be accomplished by promoting cough, giving attention to posture and using the bronchoscope. Bronchoscopic procedure in trained hands is safe, and its early use when indicated will often prevent the development of a more serious pulmonary complication.

In a series of 90 cases of postoperative atelectasis in which treatment was given by Perrone,[30] it was observed that the atelectasis occurred in 65 after cholecystostomy, cholecystectomy or gastrojejunostomy. The type of anesthetic agent employed apparently exerted no predisposing influence. Posture, the use of narcotics postoperatively and the employment of tight dressings and binders seem to be factors in the production of atelectasis. Since bronchial obstruction is the cause, every means to get rid of this should be utilized, once the diagnosis has been made. Its removal can be achieved most satisfactorily by the use of the bronchoscope. Early recognition and prompt relief are important. In the series there were 6 deaths from pneumonia, the most common complication following atelectasis.

While certain observers believe that postoperative pulmonary complications are in the majority of cases embolic in origin, Sircar and

29. Gordon, E. J.: Postoperative Pulmonary Complications: An Analysis of Etiology, Diagnosis, Prophylaxis and Treatment, Am. J. Surg. **50**:294 (Nov.) 1940.

30. Perrone, J. A.: Bronchoscopy as a Treatment of Postoperative Atelectasis: Report of Ninety Cases, Ann. Otol., Rhin. & Laryng. **49**:528 (June) 1940.

Boston [31] found that it is difficult to disassociate the responsibilities of anesthesia from those of surgical procedure in cases such as these. They discussed the importance of the preanesthetic and postanesthetic periods as well as the management of anesthesia. Certain pulmonary complications are due to the collection of secretion or foreign material in the tracheobronchial tree. Often cough and ciliary action, the normal means of removing secretion, are inadequate, and it may be necessary to resort to mechanical means for the clearing out of the secretion. Sircar and Boston recommended the use of a catheter passed through an intratracheal tube either blindly or under direct vision with the laryngoscope.

PULMONARY ABSCESS

The treatment of nontuberculous pulmonary abscess still is in an indefinite state, for there is no distinct point of demarcation in the course of the disease indicating where one plan of therapy should be abandoned and another adopted either singly or in combination. Jackson and Judd [32] reviewed a series of 124 cases of pulmonary abscess, with a view to studying particularly bronchoscopic measures as one phase of the treatment and attempting to establish the relation of such measures to the problem as a whole.

The causation in their series conformed to that generally established. In 49 cases there was a history of some form of surgical operation; in 36 this was tonsillectomy. Next in frequency as a cause was some form of respiratory infection (46 cases). That the duration of the disease before treatment is instituted has a distinct bearing on the ultimate outcome, irrespective of the plan of treatment employed, is generally admitted. The average duration in the series reported was about eleven months, the shortest period being four days and the longest, eleven years.

Apart from the value of bronchoscopy in diagnosis, there is sufficient evidence to support the belief that there is a definite place for it in the treatment of pulmonary abscess. The time element, however, is important. While there is a difference of opinion concerning this, it is important that bronchoscopic treatment is not carried past the optimum time for surgical drainage. The conservative regimen of treatment alone was carried out in 87 cases (70 per cent), although 9 patients were under observation for so short a time that their cases could scarcely be included in the final statistics. In the remaining 78 cases there were 23 deaths (29.4 per cent). Thirty-three patients were known to be cured; 5 were discharged cured but have been without a follow-up

31. Sircar, B. N., and Boston, F. K.: Postoperative Pulmonary Complications, Brit. M. J. **2**:82 (July 20) 1940.

32. Jackson, C. L., and Judd, A. R.: The Role of Bronchoscopy in the Treatment of Pulmonary Abscess, J. Thoracic Surg. **10**:179 (Dec.) 1940.

within the past year and 17 were improved at the time of their discharge back to the referring physicians. Thirty-seven (30 per cent) were referred for some form of surgical care, and of these 18 (48.6 per cent) have died of various causes. Of 19 surviving patients, only 4 have survived procedures other than external drainage. It is felt that the poor results obtained by procedures other than external drainage coincide with the experience of other investigators and justify abandonment of such procedures for the apparent operation of choice, external drainage, except when complicating conditions demand lobectomy or pneumonectomy.

Since the plan of treatment of pulmonary abscess must be determined on the basis of the individual case, no one method can be considered as "the best" when the subject is discussed as a whole. On the basis of an extensive experience, Arbuckle [33] has been impressed with the fact that in practically all cases of pulmonary abscess the duration of the disease and its severity may be diminished by proper drainage. By proper drainage is meant bronchoscopic removal of granulation tissue in the bronchial fistula, the application of suction to the contents of the cavity itself, the instillation of medicaments into the abscess cavity and postural drainage. In his experience this plan of treatment has resulted in cures in about one half of all patients with abscess free from complications.

In a series of 43 patients, 20 failed of recovery or satisfactory improvement by bronchoscopic treatment alone. Of the remaining 23, 19 made complete recovery and 4 were greatly improved. The abscesses in the 19 cured patients were acute, subacute and chronic, the shortest duration being two weeks and the longest three and one-half years. The ages of the patients varied from 3½ to 65 years.

In a study of 41 cases of abscess of the lung, Geary [34] found that aspiration, particularly during the course of anesthesia in operations around the nose and throat, was the most common etiologic factor. In 11 of the cases the abscess developed after the removal of tonsils and adenoids.

While general medical measures, including absolute rest in bed and postural drainage, are necessary, recovery is dependent on adequate drainage. This can be accomplished by the use of the bronchoscope and by external surgical drainage. The bronchoscope serves a triple purpose in treating pulmonary abscess. It is of great value in removing secretion, and certain patients respond dramatically to this form of therapy. It is of value in detecting and removing foreign material

33. Arbuckle, M. F.: Bronchoscopic Treatment of Lung Abscess, Ann. Otol., Rhin. & Laryng. **49**:821 (Sept.) 1940.

34. Geary, P.: Pulmonary Abscess: A Report of Forty-One Cases, J. M. Soc. New Jersey **37**:297 (June) 1940.

from the bronchus. It is also helpful in diagnosis, for by determining the exact bronchial branch from which the pus drains one may localize the source of the purulent secretion to a certain part of the lung. In Geary's series the bronchoscope was used in 25 of the 41 cases for diagnosis and treatment.

BRONCHIECTASIS

In a follow-up study of 400 proved cases of bronchiectasis, Perry and King [35] found that in 69 per cent the disease started in the first or second decade of life. Some statistical evidence was found to support the impression that patients in whom bronchiectasis develops before the age of 10 do not live beyond the age of 40. Of the patients with onset in the first decade, 9.4 per cent lived to or are living at the age of 40 or over. Of the 59 patients who reached the age of 40 or over, only 15 per cent had the onset of disease in the first decade.

A 12 year study of the entire group showed that the mortality among the nonsurgically treated patients was 26 per cent. Forty-one per cent of these died within five years after the onset, and 15 per cent lived 20 years or longer after the onset. Seventy-eight per cent of the patients in whom the condition proved fatal died directly as the result of this disease. For patients with the saccular type of bronchiectasis the mortality rate was 37 per cent, as compared with 13 per cent for those with the cylindric type. The operative mortality in 122 lobectomies performed on 116 patients was 3.3 per cent. While the patients who were considered the best risks undoubtedly were selected for surgical treatment, a review of those who did not have surgical treatment showed that 50 per cent had unilateral disease and 75 per cent were anatomically suitable for operation. While the mortality figures in the surgical and those in the nonsurgical group are not entirely comparable, it still is evident that lobectomy is the treatment of choice in bronchiectasis, especially in unilateral disease. Nonsurgical treatment is only palliative.

In a discussion of the prevention of bronchiectasis, Hart [36] agreed with other observers that this condition is commonly acquired, usually during childhood, and that congenital bronchiectasis is rare. There is increasing clinical and experimental evidence that atelectasis commonly precedes bronchiectasis.

Etiologically, bronchial obstruction is frequently mentioned. It is unquestionably of importance, but most likely because of the atelectasis secondarily produced and not because of primary injury to the bronchial

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35. Perry, K. M. A., and King, D. S.: Bronchiectasis: Study of Prognosis Based on Follow-Up of Four Hundred Patients, Am. Rev. Tuberc. **41**:531 (May) 1940.

36. Hart, V. K.: Prolonged Atelectasis of Both Foreign Body and Non-Foreign Body Origin: Bronchiectasis as End Result and Its Prevention by Bronchoscopic Care, South. M. J. **33**:487 (May) 1940.

wall at the site of the obstruction. Therefore, prompt bronchoscopic relief of any atelectasis is of the utmost importance if bronchiectasis is to be forestalled. This was borne out by the clinical cases cited. There was a direct relationship between early bronchoscopic treatment and the speed and completeness of cure. Similarly, these cases showed that the longer the delay of adequate drainage the longer was the period required for recovery and that prolonged neglect meant permanent dilatation of the bronchi, with ensuing chronic invalidism. Once the bronchiectasis is definitely established, there is no cure but radical surgical procedure, and this can be used only in selected cases. This fact further emphasizes the importance of prophylactic bronchoscopic care.

Myers and Blades [37] discussed the value of iodized oil in a study of certain diseases of the chest and agreed that it is indispensable in a study of bronchiectasis. Before the introduction of iodized oil for mapping of the lungs it was believed that nontuberculous bronchiectasis usually was a lesion of the lower lobe. The employment of iodized oil has shown that infection of the middle and upper lobe is not uncommon. In 57 per cent of the patients with bronchiectasis involving the lower lobe of the left lung, Myers and Blades found that the lingula of the upper lobe also was involved. Involvement of the middle lobe was demonstrated in 59 per cent of the patients with bronchiectasis involving the lower lobe of the right lung.

FUNGUS INFECTION OF THE LUNGS

The diagnosis of primary mycotic infection of the bronchopulmonary tract often is not made until postmortem observations and studies of the tissues reveal the true nature of the infection, the fungi in the sputum during life having been considered contaminants and of no clinical significance. Mycotic infections of the lungs may run a mild course with little fever and few symptoms and signs referable to the lungs or suggesting chronic bronchitis. The average case of fungus infection of the lungs resembles a case of tuberculosis and often is mistaken for it. The characteristic finding in most of these is the disproportion between the symptoms and the amount of pathologic change.

In a study of 350 patients referred to Vadala [38] for bronchoscopic investigation, routine cultures of bronchial secretion were made and examined for fungi. While the number of mycotic infections discovered was not recorded, the findings in 11 cases were recorded in detail.

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37. Myers, D. W., and Blades, B.: The Value of Proper Interpretation of Lipiodol Bronchograms in Thoracic Surgery, Am. J. Roentgenol. **44**:530 (Oct.) 1940.

38. Vadala, A. J.: Mycotic Infection of the Broncho-Pulmonary Tract, Ann. Otol., Rhin. & Laryng. **49**:291 (June) 1940.

Vadala concluded that mycotic diseases affecting the respiratory tract can be diagnosed definitely by bronchoscopic examination and study. Aspiration of exudate from the bronchi, uncontaminated by infections in the mouth and pharynx, combined with bacteriologic study is a certain method of recognizing these maladies. While proper roentgen interpretation aids immensely, final diagnosis depends on aspirating exudate through the bronchoscope and developing cultures to differentiate between tuberculosis and mycosis.

Zelman [39] reported a case in which the presence of Streptothrix in the lung presented a difficult diagnostic problem. While there were some signs of involvement within the chest, the outstanding complaints were referable to the left hip. These became progressively more marked, and surgical drainage ultimately was resorted to. The pulmonary findings were inconclusive, and no diagnosis could be arrived at. A bronchoscopic examination was performed, and secretion was secured for study. This exhibited an organism which ultimately was classified as an anaerobic streptothrix. It is of interest that iodide therapy was of no avail. The patient was eventually discharged symptom free after a course of thymol therapy.

BRONCHOSCOPY IN TUBERCULOSIS

Although tuberculosis of the tracheobronchial tree has been known for many years, its influence on the prognosis and treatment of pulmonary tuberculosis has been observed only recently, with more general employment of the bronchoscope. In a study of bronchoscopy in relation to tuberculosis, Sharp and Gorham [40] found that in many institutions it is now routine before certain major surgical procedures are carried out and is coming to be more commonly used in that manner. It is a relatively simple and harmless procedure when performed properly by trained workers, and postbronchoscopic complications are rare. Sharp and Gorham suggested that more bronchoscopic examinations be performed on patients in sanatoriums, not only to add to the knowledge of tracheobronchial tuberculosis but also with the more important aim of a more rational and better treatment of patients. There is no accepted method of treatment of tracheobronchial lesions, but a 30 per cent solution of silver nitrate locally applied seems to have some value, particularly in assisting the healing of lesions in an early stage.

While much information has been gained during the past decade by the employment of the bronchoscope in study of tuberculosis of the

39. Zelman, J.: Streptothrix of the Lung, California & West. Med. **53**:178 (Oct.) 1940.

40. Sharp, J. C., and Gorham, C. B.: Routine Bronchoscopy in Tuberculosis, Am. Rev. Tuberc. **41**:692 (June) 1940.

trachea and larger bronchi, little progress has been made in the treatment of these lesions.

In a review of the subject, Jenks [41] found that tuberculous tracheobronchitis may occur as a complication in any case of pulmonary tuberculosis, irrespective of the degree of involvement, and it may be the primary focus of active disease. While subjective symptoms, physical signs, changes in the sputum and roentgen findings may suggest the presence of tracheobronchial disease, commonly when these changes are observed the lesions are well established and usually progressive. It would seem, therefore, that bronchoscopic examination is indicated in all cases in which tuberculous tracheobronchitis is suspected. The contraindications to the use of the bronchoscope in tuberculous patients are relatively few, the most frequently mentioned being ulcerative laryngitis, recent hemorrhage, hopelessly advanced disease and extensive tracheal lesions, except when obstructive dyspnea is present. The opinion generally held is that bronchoscopic examination is not a hazardous procedure and that it promises the most aid in diagnosis as well as in treatment.

While many plans of therapy have been advocated, authorities differ as to their effectiveness and safety. Nonulcerated and nonstenotic lesions have been treated with direct ultraviolet rays applied through the bronchoscope. Ulcerated lesions are commonly treated by some form of cauterization, with the use of a 5 to 30 per cent solution of silver nitrate, electrocoagulation or a combination of these. Stenoses are treated variously. In the absence of ulceration or granulations, dilation with bougies may be practiced. Benefit is usually temporary. Ionization has been practiced, and severance of the strictures by means of the electrocautery has been attempted, but the data available do not permit one to evaluate these measures.

Continued effort must be made to find an effective plan of treatment, and bronchoscopy, performed frequently and freely, is necessary to this end. Early recognition of the complication will lessen the unfavorable results of collapse therapy.

Tuberculous hilar glandular enlargement may interfere with respiration in many ways. Simple external compression may narrow or completely stenose the bronchial lumen. A tuberculous lymph node may ulcerate through the bronchial wall and rupture into the lumen, producing acute asphyxiation. Tuberculous cicatricial stenosis of a bronchus may result, with consequent interference with drainage and respiration, and ultimately may produce extensive structural changes in the involved portion of lung.

41. Jenks, R. S.: Tuberculous Tracheobronchitis, Am. Rev. Tuberc. **41**:692 (June) 1940.

In a discussion of this subject and the mechanics which may produce the obstructing phenomena, Hughes and Simpson [42] reported 2 illustrative cases. In 1 of these there was obliteration of the lower and middle lobes of the right lung as a result of compressive stenosis. Lung mapping revealed dilatation of the involved lobar bronchi. In addition, there was marked edema of the face and upper extremities, which later involved the entire body. This, together with the bronchial stenosis, resulted from compression of the superior vena cava by enlarged tuberculous lymph nodes. In the second case there was obstructive emphysema of the right lung. At bronchoscopic examination there was found compression stenosis of the right main bronchus, and pneumonographic studies revealed marked bronchiectasis of the lower lobe.

In discussing tuberculosis of the trachea and bronchi, Myerson [43] emphasized that the most important single increase of knowledge of tuberculosis during the past ten years has come from the more general employment of bronchoscopy. The ulcerogranulomatous form of tuberculosis of the trachea and bronchus is the most common manifestation of this disease. It represents the intermediate stage of tuberculous infection, following closely on submucosal infiltration, and is transformed into a fibrotic lesion as it heals, with resulting unyielding stenosis.

The location of the lesion observed at bronchoscopic examination usually represents an extension from the point of origin rather than the actual site of beginning. It is therefore impossible in the majority of cases to see the early lesion, which commonly develops in a branch bronchus. In the cases encountered by Myerson, the common indications for bronchoscopy were:

1. As a routine procedure before thoracoplasty.
2. A positive sputum in the presence of an apparently controlled pulmonary condition, as a result of thoracoplasty or pneumothorax.
3. A spread of the disease to healthy lung which was not expected on the basis of the existing pulmonary condition.
4. A wheezing, coarse, noisy type of breathing.
5. Unexplained atelectasis.

A detailed report on 11 patients examined bronchoscopically, with the conditions observed and the treatment recommended, is included in the article.

The findings in the case reported by Feinsilver [44] emphasized the importance of bronchoscopic examination in the presence of unexplained

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42. Hughes, J. G., and Simpson, W. L.: Bronchiectasis Following Atelectasis in Tuberculosis of Infancy: Role Played by Enlarged Glands and Granulation Tissue, J. Pediat. **17**:197 (Aug.) 1940.

43. Myerson, M. C.: Tuberculous Ulcerogranuloma of the Trachea and Bronchi, Ann. Otol., Rhin. & Laryng. **49**:177 (March) 1940.

44. Feinsilver, O.: Tuberculous Bronchial Polyp, Am. Rev. Tuberc. **42**:540 (Oct.) 1940.

symptoms or physical signs referable to the chest. A smooth pedunculated tumor, which moved during respiration, was seen bronchoscopically at the orifice of the bronchus to the middle lobe. It was removed and found to contain histologic evidences of a tuberculous lesion as well as old fibrous tissue. Prior to this the patient had not been considered to have tuberculosis, as the sputum was negative for tubercle bacilli and the roentgenogram exhibited a cloudy density over the right lower part of the chest anteriorly, corresponding to the middle lobe. There was a complete absence of physical signs.

The increasing employment of bronchoscopic examination undoubtedly accounts for an increase in the frequency of the clinical diagnosis of bronchial tuberculosis. A bronchoscopic study of patients for whom various forms of collapse therapy have been unsuccessful has indicated that failure of this form of treatment often is due to endobronchial causes. Aufses [45] in a study of this subject found bronchial obstruction due either to bronchial tuberculosis, extrabronchial pressure or kinking of a bronchus as a fairly common complication of pulmonary tuberculosis. Two cases are cited in which partial bronchial obstruction exerted an unfavorable effect on collapse therapy. In 1 there was partial obstruction of the bronchus to the upper lobe of the left lung, which nullified the effects of intrapleural pneumonolysis, pneumothorax and intracostal neurectomy. In 2 cases pneumothorax was promptly followed by a tremendous increase in the size of the tuberculous cavity. Bronchoscopic examination revealed definite kinking of the bronchi leading to the cavity. After the reexpansion of the lung, the cavity returned to its original size. It was believed that this case was not suitable for collapse therapy.

While much has been written about the bronchoscopic observation of tuberculous lesions in adults, the medical literature contains relatively little about bronchoscopy in tuberculous children. During the past two years Myerson [46] examined bronchoscopically 29 tuberculous children. Their ages ranged from 5 to 15 years. The indications for bronchoscopy were: unexplained atelectasis; the need to obtain bronchial secretions when the feces and gastric secretion were negative for tubercle bacilli; the desirability of localizing the active lesion in the lung; sputum positive for tubercle bacilli in the presence of an apparently successful pneumothorax, and noisy breathing. Among the 29 children bronchoscopic examination resulted in 13 positive findings as follows: indentation by enlarged glands, 7; perforation by a tuberculous gland, 1; absorption of cartilages and collapse of a bronchial wall, 1; atelectasis due to secretion, 3; atelectasis due to ulcerogranuloma, 1.

45. Aufses, A. H.: Bronchial Obstruction and Collapse Therapy, Am. Rev. Tuberc. **42**:622 (Nov.) 1940.

46. Myerson, M. C.: Bronchoscopy in Tuberculous Children, Ann. Otol., Rhin. & Laryng. **41**:1064 (Dec.) 1940.

The ulcerogranulomatous type of lesion frequently observed in adults is not common among children. The most common gross lesion in children is produced by the pressure of enlarged mediastinal glands on the bronchial wall. Perforation into the lumen by a tuberculous process originating in an adjacent lymph node is uncommon, as also is absorption of cartilage due to external pressure.

In a bronchoscopic study of 80 patients with pulmonary tuberculosis, Laff [47] enumerated the indications for bronchoscopy as follows: symptoms of tracheal or bronchial obstruction, lobar or massive atelectasis, obstructive emphysema, unexplained hemoptysis and persistently positive sputum. In addition, bronchoscopy should be a routine procedure prior to thoracoplasty and also should be performed more frequently before induction of pneumothorax. Thirty-three of the patients exhibited tuberculous lesions involving the trachea or larger bronchi. Extensive tuberculous lesions may be present in the trachea or bronchi without apparent parenchymal disease. In Laff's experience no unfavorable developments followed bronchoscopic examination.

Frequently bronchoscopic inspection has resulted in the recognition of unusual forms of tuberculosis involving the tracheobronchial tree. Compression of a bronchus by a tuberculous lymph node and ulceration into the bronchus of so-called "lung stones" have been reported. Ulceration into the lumen of a bronchus by an actively diseased tuberculous lymph node with little or no calcification is rare.

In the case reported by Vinson and Pembleton [48] a tumor occluded the bronchus of the lower lobe of the right lung in a man aged 68 years. For two weeks there had been an irritative cough, which culminated in a copious hemorrhage. At bronchoscopy it was possible to remove a large mass of tissue, which proved to be a large anthracotic lymph node. Microscopic evidences of tuberculosis were observed. The patient made a prompt recovery.

BRONCHOLITHIASIS

The migration of calcifications from a lung or a lymph node into the bronchial lumen is occasionally observed. A positive diagnosis can be made only on bronchoscopic visualization of the broncholith or on observation of it in the sputum.

In 7 cases observed by Myers [49] it seemed reasonably certain that a broncholith was the cause of pulmonary hemorrhage. In 4, positive confirmation was obtained, once at necropsy, once in an operative speci-

47. Laff, H. I.: The Value of Bronchoscopy in Pulmonary Tuberculosis, Rocky Mountain M. J. **37**:662 (Sept.) 1940.

48. Vinson, P. P., and Pembleton, W. E.: Ulceration of Tuberculous Hilar Lymph Node into Lumen of Bronchus with Bronchoscopic Removal: Report of a Case, Ann. Otol., Rhin. & Laryng. **49**:797 (Sept.) 1940.

49. Myers, D. W.: Broncholithiasis, Dis. of Chest **6**:269 (Sept.) 1940.

men, when lobectomy had been successfully performed for uncontrollable hemorrhage, and twice by bronchoscopy, when a broncholith was removed and evidences of erosion or ulceration of the bronchial wall were observed. In the remaining 3 cases calcareous material was expectorated and studies failed to reveal any cause for the hemoptysis.

DISEASES OF THE ESOPHAGUS

In a general discussion of diseases of the esophagus Clerf [50] reemphasized the importance of giving careful consideration to symptoms of early esophageal disease, notably to dysphagia. Often great difficulty is experienced in the proper interpretation of vague sensations experienced by the patient when swallowing food or saliva, and unless one is sympathetic to these early manifestations of disease, errors in diagnosis will result. It is important not to ascribe them to a supposedly functional disorder.

Among methods of diagnosis a study of the swallowing function by roentgen examination should not be omitted. In cases of doubt and for purposes of corroboration of a diagnosis, the use of the esophagoscope is indicated. A number of esophageal diseases were discussed from a diagnostic standpoint, and treatment was mentioned.

Congenital Atresia.—In discussing the subject of congenital esophageal atresia, Lanman [51] emphasized the importance of the symptoms, which are characteristic and striking namely, attacks of choking and cyanosis in the first few hours of life. These invariably are aggravated whenever fluid is given, because of the aspiration of materials into the air passages. It is noted that between attacks the child has an excessive quantity of secretions in its mouth and throat.

He recommended that a roentgenogram should be made without the use of a contrast medium and advised against the employment of barium sulfate or other substance, because of the hazards incident to aspiration of these into the air passages.

In discussing certain pathologic conditions involving the esophagus, Clerf [52] emphasized that physicians should take a more sympathetic attitude toward patients who complain of esophageal symptoms, irrespective of the apparently trivial or absurd character of the symptoms. Dysphagia is most often ignored or misinterpreted. Careful and complete examination of the upper air and food passages should be made. Palpation

50. Clerf, L. H.: Diseases of the Esophagus: Esophagoscopic Considerations, Arch. Surg. **41**:1043 (Nov.) 1940.

51. Lanman, T. M.: Congenital Atresia of the Esophagus: A Study of Thirty-Two Cases, Arch. Surg. **41**:1060 (Nov.) 1940.

52. Clerf, L. H.: Pathologic Conditions of the Esophagus, Laryngoscope **50**: 463 (May) 1940.

of the neck should be a routine procedure. Roentgen study of the swallowing function should be done if the symptoms or signs cannot be satisfactorily explained by physical examination. Esophagoscopic examination is indicated in all cases of esophageal disease requiring corroboration of the roentgen findings. It also is indicated in the absence of roentgen evidence if there is a reasonable belief that disease of the esophagus is present. It is important to bear in mind that in the practice of medicine errors by omission are more common than errors by commission.

Plummer-Vinson Syndrome.—Kernan [53] reviewed the literature and discussed the symptoms and pathologic evidences of the Plummer-Vinson syndrome. These include anemia, dysphagia, glossitis and achlorhydria. On the basis of the views expressed, dysphagia may be caused by (1) spasm due either to hysteria or to atrophic or painful lesions which lead to overstimulation or to understimulation of the sensory arc of the swallowing reflex or (2) actual mechanical obstruction caused by bands or webs across the mouth of the esophagus.

Other obvious questions presented themselves, as follows: What is the relation between the dysphagia and the anemia? Is there a relation between achlorhydric anemia and the Plummer-Vinson syndrome? Is there any relation between gastric ulcer and this syndrome or between the syndrome and carcinoma? Are all the symptoms different phases of one disease, for which some toxic or dietetic factor is responsible? And finally, what is the causative factor? Kernan indicated that as yet no clear answer has been given to any of these questions. From the reported cases one may conclude that the syndrome rarely presents itself as a whole or that several different conditions have been described.

In 2 cases reported by Kernan, which were not considered as instances of the Plummer-Vinson syndrome, there was a long history of gastric disease, surgical operation on the stomach and probably also dietary deficiencies. At esophagoscopic examination numerous weblike adhesions were found throughout the greater part of the esophagus. Both cases terminated fatally. Kernan expressed the opinion that many conditions characterized by dysphagia, anemia and glossitis and reported as examples of the Plummer-Vinson syndrome must represent some other entity which cannot be explained on a hysterical basis.

The association of hypochromic anemia and dysphagia has interested many laryngologists, since the dysphagia is so common in many of the patients, who are first examined by laryngologists. Roentgen and esophagoscopic examination of the pharynx and esophagus in cases such as this generally have been thought to be of minor importance.

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53. Kernan, J. D.: Plummer-Vinson Syndrome: Report of Two Cases, Arch. Otolaryng. **32**:662 (Oct.) 1940.

In Gerlings'[54] 6 patients suffering from dysphagia and anemia, roentgen examination revealed one or another of the following: stagnation in the vallecula and in the piriform sinus, stagnation of barium sulfate above the mouth of the esophagus, spastic stricture immediately below the mouth of the esophagus and cicatricial stenosis of the mouth of the esophagus or of the esophagus itself. On 5 of the 6, esophagoscopic examination was performed. In 2 there were found disorders of the mucosa. In one this was associated with spasm, while a distinct area of leukoplakia with fissures was noted in the other. In 3 there was erosion of the mucosa of the upper part of the esophagus, with some stenosis. In 2 of these the process was subsiding, while in the third severe cicatricial stenosis had developed, which necessitated dilation of the stenotic area. Stenotic changes may be demonstrated by roentgen examination, and this study should be performed before any instrumentation of the esophagus is attempted.

There still is considerable controversy as to the connection between the atrophic changes of the mucosa and hypochromic anemia. The fact that carcinoma often develops secondarily to these changes makes esophagoscopic examination imperative. While certain writers deny that this occurs, it must be remembered that the mucosal changes and other evidences clear up if appropriate treatment is instituted, whereas in the untreated patient there is a tendency for the process to become progressively worse, and it seems that these untreated patients are predestined for carcinoma.

Excellent roentgenographic illustrations of the changes noted in the piriform sinuses and about the upper orifice of the esophagus accompany the article.

In an experimental study to determine the effects of anemia on the esophagus, Cordray[55] placed 12 rats on a diet consisting entirely of milk. At the end of five and one-half months the rodents were weak and underweight and the fur was very coarse and thin. Studies of the blood showed profound anemia. Microscopic examination of slides of the esophagus from each of the 12 rats revealed the following:

> . . . the epithelium of each rat in the group of 12 showed marked hyperkeratinization with proliferation of the cells comprising the epithelial layer and increase in the number of mitotic figures over the usual normal number. This points to a precancerous type of lesion. No fibrous tissue replacement in the musculature was evident, thus conclusively ruling out that web formation might arise as a result of anemia. Just below the basement membrane, in various sections, were detected epithelial cells which had apparently broken through this membrane.

54. Gerlings, P. J.: Disorders of the Mouth of the Oesophagus in the Syndrome of Plummer-Vinson, J. Laryng. & Otol. **55**:143 (March) 1940.

55. Cordray, D. P.: The Plummer-Vinson Syndrome, Ann. Otol., Rhin. & Laryng. **49**:160 (March) 1940.

The author concluded that severe secondary anemia in no way predisposed to web formation and that in the Plummer-Vinson syndrome the web is the cause of the anemia, forming a barrier to adequate nutrition.

TUMORS OF THE ESOPHAGUS

Fibroma.—While benign tumors of the esophagus can no longer be considered as pathologic curiosities, their occurrence is not common. In a study of this subject, Barrett [56] found that those arising from the mucosa or submucosa, which comprise the majority of benign tumors, are sessile or pedunculated. He reported a fibroma of unusual size occurring in a woman aged 66 years. The symptoms complained of were cough, dyspnea and wheezing. There were no symptoms referable to the esophagus, and there was no loss of weight. The physical signs and roentgen evidences were those characteristic of a superior mediastinal mass. No esophagoscopic examination was carried out. A thoracotomy was performed, and the tumor was found to form part of the wall of the esophagus. Esophagectomy was indicated but was not warranted because of the patient's age and condition. At autopsy the tumor was found growing from the wall of the esophagus. The pathologic diagnosis was fibroma. Excellent roentgenograms and photographs of the specimen accompany the article.

The occurrence of true benign polypoid tumors in the esophagus is unusual. Two cases were reported by Mahoney.[57] Esophagoscopic examination in 1 revealed a firm mass at the aortic arch; a second similar but lobulated lesion was observed near the lower end of the esophagus. The histologic diagnosis was esophageal polypi. The appearance of the lesion remained practically unchanged when it was observed almost three years later. In another case several biopsies revealed polyps, corroborating the esophagoscopic diagnosis, but subsequently a diagnosis of cancer was made.

Carcinoma.—While a positive diagnosis of achalasia of the cardia usually can be made on the basis of the history and roentgen findings, Kornblum and Fisher [58] directed attention to the need of careful study of the findings and often of esophagoscopic examination also. Three cases are reported in which carcinoma of the esophagus was found. One of these was an instance of carcinoma of the cardia that simulated achalasia. The diagnosis of carcinoma was made after esophagoscopic inspection. In the remaining 2 cases carcinoma was superimposed on a

56. Barrett, N. R.: Fibroma of the Esophagus: Report of a Case, J. Thoracic Surg. **9**:672 (Aug.) 1940.

57. Mahoney, J. J.: Polypoid Tumors of the Esophagus: Report of Two Cases, Laryngoscope **50**:1096 (Nov.) 1940.

58. Kornblum, K., and Fisher, L. C.: Carcinoma as Complication of Achalasia of Cardia, Am. J. Roentgenol. **43**:364 (March) 1940.

previously diagnosed achalasia and was recognized only by direct examination. Kornblum and Fisher expressed the opinion that no investigation of achalasia of the cardia can be considered complete without direct visualization through the esophagoscope.

The common malignant tumor of the esophagus is carcinoma. Sarcoma is rare, and rarer still are those tumors of uncertain origin and histology which have been designated as carcinosarcoma, sarcocarcinoma or carcinoma sarcomatodes. In a comprehensive study of the world's literature on carcinosarcoma of the esophagus and of 8 cases of carcinoma seen at autopsy, Pearlman [59] expressed the opinion that the presence of so-called carcinosarcoma in the esophagus or elsewhere is questionable. He analyzed and discussed the possible sources of error leading to such a diagnosis.

Blastomycosis.—Although several hundred cases of blastomycosis have been reported, in only 3 has there been involvement of the esophagus. Clinically, two types of the disease have been described, a cutaneous and a systemic type. In the cutaneous type the lesions are few and appear on the exposed surfaces of the body, while in the systemic type several multiple lesions are usually present, with involvement of many organs and structures. Lesions of the gastrointestinal tract are rare. The diagnosis can best be made by biopsy or by examination of a smear or by culture of the organism. The disease must be differentiated from other mycoses, tuberculosis, syphilis and malignant growth. In the esophagus it resembles carcinoma both clinically and roentgenologically.

In the case reported by Straus,[60] that of a man aged 61, there was a history of progressive dysphagia of two years' duration. Lesions in the nose and lower lip were diagnosed by biopsy as blastomycosis. Roentgen study of the swallowing function revealed almost complete obstruction of the esophagus in its upper third. At esophagoscopic examination there was found a soft granular constricting lesion which was annular and was located 6 cm. below the cricopharyngeus muscle. Slight trauma produced bleeding. Biopsy revealed chronic inflammatory tissue. A second biopsy demonstrated inflammatory tissue in which were found typical blastomycetic organisms. Smears and cultures of material taken directly from the lesion revealed no organisms. Examination of the larynx, trachea and bronchi revealed no abnormalities, and the secretions were normal. The patient failed to respond satisfactorily, and death ensued.

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59. Pearlman, S. J.: So-Called Carcino-Sarcoma of the Esophagus, Ann. Otol., Rhin. & Laryng. **49**:805 (Sept.) 1940.

60. Straus, G. D.: Blastomycosis of the Esophagus, Arch. Otolaryng. **32**:1082 (Dec.) 1940.

At autopsy the upper end of the esophagus was found to be the only site of disease. The lesion appeared as a large annular mass, which exhibited a crater-like ulcer at its upper part and complete erosion of the mucosa at its lower end. Between these were several small raised tubercle-like structures. The remainder of the esophagus was normal.

Tuberculous Periesophageal Abscess.—Primary tuberculous involvement of the esophageal wall is rare. Usually this site is involved secondarily in extension of a tuberculous process in mediastinal lymph nodes, bronchi or lungs or as an adhesion between a tuberculous lymph node and the esophageal wall.

In the case reported by Clerf [61] a man aged 22 years had a rather abrupt onset of dysphagia and pain in the chest. Roentgen study of the swallowing function revealed stenosis and deformity. At esophagoscopic examination an abscess was found draining into the esophagus. Tubercle bacilli were found in the pus. The patient made an uneventful recovery. In the absence of any other demonstrable focus of tuberculosis it was concluded that this was a case of tuberculosis of a lymph node which had ruptured into the esophagus.

In a discussion of the various forms of treatment of esophageal varices, Walters and others [62] reported the recent successful obliteration of varices in a series of 6 cases by the injection of a sclerosing solution through the esophagoscope. A solution of 2.5 per cent sodium morrhuate was injected through a special 25 caliber needle into a suitable vein. A detailed report of the 6 cases is included in the article. While sufficient time has not elapsed in which to determine the efficacy of the injection method of treating esophageal varices, the method nevertheless is worthy of a trial, and future reports will be awaited with interest.

FOREIGN BODIES IN AIR AND FOOD PASSAGES

In compiling data covering end results in cases of foreign body many difficulties present themselves. Complications and immediate end results may be recorded, but accurate data on sequelae, particularly in cases of bronchial foreign bodies of long sojourn, are difficult to secure. The reasons are the wide geographic distribution of patients, difficulty in evaluating data submitted and failure to reach patients by the accepted follow-up methods.

A series of 950 consecutive cases in which foreign bodies were removed by endoscopic means from the air and food passages was reviewed

61. Clerf, L. H.: Tuberculous Periesophageal Abscess Producing Stenosis: Report of a Case, Ann. Otol., Rhin. & Laryng. **49**:793 (Sept.) 1940.

62. Walters, W.; Moersch, H. J., and McKinnon, D. A.: Bleeding Esophageal Varices: An Evaluation of the Methods Directed Toward Their Control, Especially by Direct Injection of a Sclerosing Solution, Arch. Surg. **41**:1101 (Nov.) 1940.

by Clerf [63] from the standpoint of postoperative complications, deaths and sequelae. In 413 cases the foreign bodies were in the air passages, and in 537, in the food passages. Nuts, seeds and shells were the foreign bodies in 215 cases, or over 22 per cent. Of these, 115 were peanuts. Bones ranked next to vegetal foreign bodies, being found in 166 cases, in 19 of which they were in the air passages and in 147 in the food passages.

Dyspnea occurred more often, and tracheotomy was required more frequently, for foreign bodies of vegetal origin. Subcutaneous emphysema of the neck was observed not infrequently after tracheotomy. In 3 cases of foreign body in the bronchus subcutaneous and mediastinal emphysema was observed before any instrumentation was performed.

Pulmonary abscess, an uncommon complication of bronchial foreign body, was observed in only 1 case. Bronchiectasis, however, was observed often, particularly in cases in which a foreign body had been present for a long time and in complete bronchial obstruction with suppuration.

Unfavorable developments following the lodgment of a foreign body in the esophagus were not as common as in cases in which the foreign body was in the air passages. The common complications were injury to the esophageal wall, with infection, varying from traumatic esophagitis to mediastinitis. Fatal results also occurred from pressure necrosis and from erosion into a large vessel or perforation of its wall. In the series of 950 cases there were 16 deaths (1.68 per cent). Twelve of these occurred in cases of foreign body in the air passages and 4 resulted from the presence of a foreign body in the esophagus. A detailed history of the cases in which the outcome was fatal is included in the article.

In presenting their experiences in a series of 206 cases of foreign body in an air or a food passage, Mahoney and Agar [64] stressed the importance of prevention. Carelessness and neglect were found to be common predisposing etiologic factors. The symptoms and diagnostic signs, and special reference to obstructive emphysema and atelectasis, were discussed.

Seydell [65] reported the case of a patient who complained of severe cough and dyspnea and in whom the presence of a foreign body in the esophagus was suspected. There was a history of severe choking while

63. Clerf, L. H.: Foreign Bodies in the Air and Food Passages: Observations on End Results in a Series of Nine Hundred Fifty Cases, Surg., Gynec. & Obst. **70**:328 (Feb., no. 2A) 1940.

64. Mahoney, P. L., and Agar, J. S.: Foreign Bodies in the Food and Air Passages, J. Arkansas M. Soc. **37**:140 (Dec.) 1940.

65. Seydell, E. M.: Center of Ossification of the Sternum Mistaken for Foreign Body in the Esophagus, Ann. Otol., Rhin. & Laryng. **41**:1061 (Dec.) 1940.

the child was playing with a celluloid toy. On several examinations roentgen studies revealed an annular-shaped object, about the size of a quarter, below the sternoclavicular joint and slightly to the left of the median line. The appearance suggested a thin metallic object or something of a similar composition. Direct examination of the larynx, trachea and esophagus did not reveal the presence of a foreign body. Examination of roentgen films of children of a similar age revealed similar disk-shaped shadows in a number of cases, and it became obvious that the shadow which had been mistaken for that of a foreign body was in reality a germinal center of the sternum. In a discussion of the case the author pointed out that ossification of the manubrium begins in a single center, although occasionally accessory centers are added. The body usually ossifies from seven centers. The upper segment ossifies from a single nucleus, and below this three pairs of nuclei appear, which may remain separate for a considerable length of time. The fact that the process may be irregular and that these centers may remain separate for a long time should suggest to roentgenologists the importance of distinguishing between these and shadows cast by foreign bodies in either the trachea or the esophagus.

Pulmonary interstitial and mediastinal emphysema is not a common complication of cases of foreign body in the bronchus. In the case reported by Fisher and Macklin [66] a girl aged 22 months aspirated a number of fragments of peanut. A few were removed bronchoscopically from the lower lobe of the right lung, which previously had shown roentgen and clinical evidences of atelectasis. Tracheotomy was subsequently performed for relief of dyspnea, but little improvement was noted. The patient died five days after the accident and three days after the bronchoscopic removal of a number of fragments of nut. At necropsy there was found marked interstitial emphysema of the left lung and of the hilar region of each lung, together with mediastinal emphysema. This apparently caused compression and obstruction of the pulmonary and mediastinal circulation, producing severe dyspnea, anoxemia and cyanosis as well as impeding the action of the heart. The principal cause of death was circulatory embarrassment from the presence of the emphysema.

According to available statistics, perforation of the esophagus with septic mediastinitis is not a common complication of esophageal foreign body. Vistreich [67] reported 2 cases of bone in the esophagus, in which there was evidence of suppuration. In one the foreign body, a chicken

66. Fisher, J. H., and Macklin, C. C.: Pulmonic Interstitial and Mediastinal Emphysema, Am. J. Dis. Child. **60**:102 (July) 1940.

67. Vistreich, F.: Two Cases of Esophageal Foreign Body with Complications, Laryngoscope **50**:1178 (Dec.) 1940.

bone, had been present in the cervical esophagus for five days. A pointed bone, which had penetrated the esophageal wall, together with a quantity of foul pus, was removed esophagoscopically. The patient made an uneventful recovery. In the second case the bone, swallowed three days before, apparently had perforated the esophageal wall. The foreign body could not be found at esophagoscopic examination. There were cellulitis of the neck, substernal pain, marked dysphagia and high fever. External drainage was not performed. At autopsy both extremities of the bone were found to have perforated the esophageal wall. These areas were surrounded by hemorrhage, suppuration and necrosis.

The prognosis in cases such as these would be more favorable if esophagoscopic removal of the foreign body could be done promptly instead of after a wait of a number of days. In the event that perforation of the esophagus and mediastinitis have occurred, the progress of the condition will not be materially influenced by esophagoscopic removal of the foreign body.

Esophagobronchial Fistula.—An unusual case, in which esophagobronchial fistula resulted from the lodgment of a bone in the esophagus and in which there was subsequent penetration of the left main bronchus, was reported by Murtagh and Tyson.[68] The foreign body accident had occurred twenty-five days previously and was followed by substernal pain, which was accentuated by swallowing. A roentgen study made five days after the accident did not reveal evidence of a foreign body. Twenty-five days later, when the patient was admitted to the hospital for study, there was no roentgen evidence of esophageal obstruction. However, during the barium sulfate study a small quantity of the injected material was seen to project forward and laterally from the esophagus to the left lung, and coincident with this the patient experienced a severe paroxysm of coughing. Esophagoscopic and bronchoscopic examinations were performed. At the crossing of the left main bronchus was found an area of ulceration with inflammation of the esophagus, and on the posterior wall of the left main bronchus, 1.5 cm. from the carina, an area of marked inflammation and ulceration. No foreign body was observed.

Gastrostomy was performed and the fistula studied further with iodized oil. Later another esophagoscopic examination was performed, and a small spicule of bone was found protruding into the esophageal lumen from the fistula. This was removed. After this there was a complete disappearance of substernal pain, the fistula healed promptly and the patient made a complete recovery.

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68. Murtagh, J. A., and Tyson, M. D.: Esophagobronchial Fistula: Result of Foreign Body; Report of Case, New England J. Med. **222**:494 (March 21) 1940.

GASTROSCOPY

Of 200 patients examined both by roentgen ray and the flexible gastroscope, Wirts [69] found a gastric lesion in 128. Since a diagnosis of gastritis cannot often be made by roentgen study, a better comparison of the value of these two methods of examination can be made if cases of gastritis are excluded. When this was done, it was found that the roentgen and gastroscopic findings were in agreement in 87 per cent of all cases. It is of interest that of 6 cases of polyps of the stomach, 1 was diagnosed by both methods and 5 by gastroscopy alone. Of 42 cases of cancer, 34 were diagnosed by both methods, 5 by roentgen examination and not by gastroscopy and 3 by gastroscopy but not by roentgen study. In 29 cases of gastric ulcer, 18 were diagnosed by both methods, while in 5 roentgen study gave positive evidence and the results of gastroscopic inspection were negative and in 6 the diagnosis was made by gastroscopy and not by roentgen examination.

The value of gastroscopy is demonstrated by the fact that it is the most definite method whereby the diagnosis of gastritis and of benign tumor can be made. In cases of ulcer or of malignant disease gastroscopy is a valuable adjunct to roentgen examination.

All observers are agreed that there are so-called "blind areas" in the stomach, which usually cannot be seen at gastroscopy. The most certain criterion for a complete observation of the stomach is visualization of the pylorus. The observation of the pylorus will convince the observer that he has seen everything that can be observed gastroscopically. Of a series of 313 cases reported by Schindler and Morales [70] the pylorus was observed in 285 (91.1 per cent). The technic for the finding of the pylorus was described.

Clinicians are constantly confronted with patients who complain of distress in the upper part of the abdomen, which conforms to no recognized symptom complex. Studies give negative results. Patients of this type are usually classified as having a functional disturbance or a gastric neurosis. Recently, attention has been directed to the frequency with which chronic gastritis has been observed in patients such as these. As a result of an analysis of 543 cases in which gastric examinations were performed on patients who complained of indefinite distress in the upper part of the abdomen and in which these studies gave negative results, Ruffin and Brown [71] concluded that the symptoms of such

69. Wirts, C. W., Jr.: An Analysis of Two Hundred Patients Examined Gastroscopically, Ann. Int. Med. **14**:583 (Oct.) 1940.

70. Schindler, R., and Morales, F. H.: Gastroscopic Inspection of Pylorus, Am. J. Digest. Dis. **7**:242 (June) 1940.

71. Ruffin, J. M., and Brown, I. W., Jr.: Occurrence of Gastritis as Diagnosed by Gastroscopy in Gastric Neuroses, Am. J. Digest. Dis. **7**:414 (Oct.) 1940.

patients, who are commonly classified as having gastric neuroses, cannot be explained on the basis of chronic gastritis. Hemorrhagic or pigmented spots were observed in 25 per cent, but their significance could not be determined.

Gastric symptoms developing after operations on the stomach often present difficult diagnostic problems. This is due to the inconsistency of the symptoms and particularly to the difficulty of roentgenographic interpretation in view of the disturbed anatomy of the stomach. In a survey of the gastroscopic findings in 100 patients on whom gastric operations had been performed and in whom distress developed subsequently, Moersch and Walters [72] found no gastroscopic evidence of disease in 30 cases. In 56 cases a diagnosis of gastritis of some type was made from the gastroscopic findings. It was observed that in those cases in which no abnormality of the gastric mucosa was demonstrated, the functional factor was pronounced and that in most instances the patients responded to therapeutic measures directed to the restoration of normal function. The response to medical management was not so satisfactory in the patients with gastritis, and many required further operation.

Other lesions, such as gastrojejunal ulcer, carcinoma, gastric ulcer and benign tumor, develop in the stomach after operation, and gastroscopy may be of assistance in their recognition. In 5 cases in the series of Moersch and Walters, carcinoma of the stomach was found on gastroscopic examination, and in 6 cases gastrojejunal ulcer was visualized.

The effect of inflation of the stomach on the gastric picture has been questioned by many observers. By some it has been noted that not only normal folds but also hypertrophic folds can be diminished in size or effaced. In a study carried out by Ruffin and Brown [73] it was noted that inflation exerted a definite effect on the size and appearance of gastric folds. Folds that seemed large, swollen and inflamed when first observed appeared practically normal after slight inflation and usually could be completely obliterated by further inflation. In certain instances folds which appeared normal disappeared under inflation and a typical picture of "atrophic gastritis" was left, with a network of blood vessels becoming visible. Ruffin and Brown concluded that inflation of the stomach effected marked alterations in the gastric picture. Hypertrophic folds were effaced by inflation. In dogs a typical picture of gastritis

72. Moersch, H. J., and Walters, W.: Gastroscopic Observations in Cases of Gastric Distress Following Operations on the Stomach, Surg., Gynec. & Obst. **71**: 129 (Aug.) 1940.

73. Ruffin, J. M., and Brown, I. W., Jr.: Effect of Inflation of Stomach on Gastric Picture, Am. J. Digest. Dis. **7**:418 (Oct.) 1940.

was produced at will. In some instances apparently normal folds disappeared under pressure and blood vessels were seen.

Gastroscopy is now a generally accepted method of examining the stomach. It bears much the same relation to gastroenterology that diagnostic cystoscopy bears to urology. In connection with a discussion of the indications for gastroscopy, Benedict [74] presented 12 illustrative cases. The first indication is gastritis. Since the finer changes of the gastric mucosa can be observed readily by means of the gastroscope, the value of this method in the diagnosis of gastritis is self evident. The second important indication is unexplained gastrointestinal hemorrhage. The procedure also is of value in the study of cases of gastric neurosis, since these often represent cases of gastritis. It is indicated also in cases in which roentgen examination gives negative results but gastrointestinal symptoms persist or in which the results of roentgen examination are inconclusive.

The procedure is of value in cases of gastric ulcer, both for diagnosis and the determination of appearances and localization and also for examination following the healing process. In certain cases of malignant disease gastroscopy is helpful in determining the extent and the operability of the lesion. It is also of value in polyposis of the stomach, in differentiating polyps from foreign bodies and in postoperative examination of the stomach, particularly in the presence of unexplained symptoms.

In support of the conviction that the use of the flexible tube gastroscope is a safe procedure in trained hands, the results of a questionnaire have been reported by Schindler.[75] Sixty gastroscopists reported their experiences during 22,351 gastroscopic examinations. In this series there was 1 death which probably can be attributed to the use of the gastroscope. Eight gastric perforations and 1 perforation of the jejunum in a resected stomach were observed. The patients recovered either after conservative treatment or after surgical intervention.

1530 Locust Street.

74. Benedict, E. B.: Indications for Gastroscopy, New England J. Med. **223**: 925 (Dec. 5) 1940.

75. Schindler, R.: Results of the Questionnaire on Fatalities in Gastroscopy, Am. J. Digest. Dis. **7**:293 (July) 1940.

Abstracts from Current Literature

Ear

EXPERIMENTAL STUDIES ON ENCHONDRAL BONE OF THE OTIC CAPSULE. W. P. COVELL, Ann. Otol., Rhin. & Laryng. **48**:934 (Dec.) 1939.

Covell reports experiments on rabbits, in which small fragments of bone from the region of the margin of the oval window were transplanted into the anterior chambers of the eyes of other rabbits and changes in the transplants were observed over a period of seven months. It was found that after seven to ten days the graft began to grow and that after thirty-three days vascular and fibrous tissue connections to the transplant were well established. There was evidence of necrosis of the old bone and of formation of new bone. The endosteal layer did not form new bone, as did the periosteal layer.

M. V. MILLER, Philadelphia.

HEARING AIDS—TUBE OR CARBON? A. A. HAYDEN, J. A. M. A. **115**:191 (July 20) 1940.

The late Dr. Hayden discusses in moderate detail, but with exactness and precision, the various types of hearing aids, tracing the development and refinement of the modern electric hearing aid and the modern radio receiving set from 1922 to the present.

Several charts augment the text. The conclusions of this paper are as follows:

"1. At present both tube and carbon aids have definite advantages and disadvantages. Both are being improved constantly.

"2. Indications for each must be formulated from the experience of otologists, physicists and manufacturers, with the final decision made by the patient.

"3. Tube aids give more brilliant hearing improvement in conduction loss when nerve function is good. In severe perceptive loss, results are often disappointing.

"4. Purchase from a firm that manufactures both carbon and tube aids, for air as well as bone conduction, allows choice between the two and makes exchange of one for the other easier.

"5. Since tube aids are much more complex instruments, the need for an impartial evaluating agency such as the Council on Physical Therapy of the American Medical Association, to set and maintain requirements for acceptance of standards of construction and performance as well as of sales ethics and advertising, is even more necessary with the advent of tube aids. Financial rating of the manufacturers is very important.

"6. Although the lure of radio tubes is great, patients should not discard carbon for tube aids without careful investigation.

"7. Tube aids are as yet more expensive to manufacture, buy, keep up and service.

"8. Whether tube aids will supplant the carbon type of hearing aid or vice versa, or whether each will eventually occupy its own definite field, with the actual improvements that are being made in construction by manufacturers and in the prescription of hearing aids by otologists, good results are now obtained by the vast majority of patients properly fitted with a well made hearing aid.

"9. Even better instruments and better hearing seem to be on the way."

GORDON, Philadelphia.

STUDIES OF THE EIGHTH CRANIAL NERVE OF MAN. A. T. RASMUSSEN, Laryngoscope **50**:67 (Jan.) 1940.

Rasmussen counted the nerve fibers in 37 normal vestibular nerves and 40 normal cochlear nerves from persons with apparently normal hearing whose ages ranged from 2 to 60. This was prompted by the discrepancy between the number of ganglion cells in the spiral ganglion and the supposed number of fibers in the cochlear division of the eighth nerve.

Because of the close association of the cochlear and the vestibular fibers throughout most of their course, it was assumed that the only way to get a reasonably accurate determination of the number of cochlear fibers was to count the nerve fibers in the intrapontile portion of the vestibular nerve and deduct the resulting figure from the total number of fibers in the extramedullary portion of the whole eighth nerve.

A method of dissecting the vestibular nerve with its ascending and descending branches within the pons and the medulla oblongata is described and illustrated. The counting procedure and the fixing and staining methods are described in detail.

Rasmussen found that the funicular pattern of the eighth nerve varies greatly in different persons and in different regions of the same nerve. When two distinct trunks are present, one is probably wholly vestibular, but the other is cochlear with a variable number of vestibular fibers. The cross section area of the vestibular portion is usually the larger, but in about one fifth of the subjects the cochlear was distinctly the larger, with the average cross section area of the entire series the same for the two components.

Apparently normal cochlear nerves contain from 22,800 to 40,000 nerve fibers, the average being 31,400. These figures are distinctly higher than those of any fiber counts on human material thus far discovered in the literature. The close agreement between these data and the independent figures on the number of spiral ganglion cells makes it safe to regard the number of conductors carrying auditory impulses into the human brain under normal conditions as about 23,000 to 40,000. The average number of cochlear fibers in man appears to be between 31,000 and 32,000, compared with nearly 50,000 in the cat. The number of vestibular fibers within the whole group varied from 14,200 to 24,000, averaging 18,500. The cochlear nerves of the older subjects contained 2,200 fewer fibers and the vestibular nerves 1,000 fewer fibers than those of the younger subjects.

WOOD, Newark, N. J.

FUNCTION OF THE OTOLITHIC ORGANS. G. TENAGLIA, Oto-rhino-laryng. internat. **23**:573 (Nov.) 1939.

Study of the otolithic apparatus, a direct descendant of the primitive otocyst, becomes more difficult in the higher animals because of the necessity for abolishing interference from the other functions that have been grafted on it. Of the methods adopted for this purpose, that of McNally and Tait, of severing the individual vestibular branches in the frog, is the most rational.

The most striking physical characteristic of the otolith is its high specific gravity. Consequently, otolithic reflexes occurring in response to a stimulus are due principally to the force of gravity. They are represented by compensatory ocular movements, labyrinthine righting reflexes, reflexes of static adaptation, tonic labyrinthine reflexes and reflexes to linear acceleration and to free falling. More recent studies indicate that compensatory ocular movements are dependent on the utricle, since they persist after abolishment of the saccular organ. Labyrinthine righting reflexes are also of utricular origin; after unilateral labyrinthectomy they are manifest only when the animal is in a lateral position, on the side opposite to that operated on. The reflexes of static adaptation and the tonic labyrinthine reflexes are especially important, because they principally assure the maintenance of equilibrium; they are also of utricular origin. Reactions to linear acceleration are in relation to the force of gravity and to the force of inertia.

The reactions are more evident in terrestrial, horizontal acceleration than in flying. In the latter, the force of the stimulus is weaker because of the uniformity of speed. Climbing, rapid descent and the performance of acrobatics produce strong stimuli; wide turns cause little angular acceleration, and in looping, the angular acceleration is below the perceptible minimum, so that the semicircular canals are not stimulated, although centrifugal action and the force of gravity stimulate the otoliths. During tight spirals and spinning, both the canals and the otoliths are stimulated. Other functions contribute to the regulation of flight, notably vision and the superficial and the deep sensibility. The righting reaction in free falling is abolished after bilateral labyrinthectomy. Probably vision and the otolithic and canalicular reflexes cooperate in the righting reflex. It cannot be denied that labyrinthine excitations cause neurovegetative manifestations, but these cannot be attributed solely to the excitations.

Many experimental findings point to the presence of a hearing function in the posterior division of the labyrinth, although the question is not yet resolved. To each pair of otoliths has been attributed a special function. Thus, the lapilli are said to control the flexor and extensor muscles of the neck, trunk and limbs and vertical movements of the eyes, while control of the rotatory ocular movements and of the abductor and adductor muscles is under the dominion of the sagittae. Recent studies, however, seem to exclude the saccule from participation in the maintenance of equilibrium. There has been much discussion of the manner of action of stimuli on the sensorial elements. It can be stated only that the predominant stimulus is the force of gravity and this observation lies at the base of every one of the theories, whether gravity is conceived to act directly by traction, pressure or bending of the cilia or by electric stimulation, osmosis or hydrostatic pressure. It should be remembered that the various divisions of the labyrinth really constitute a functional complex, and when one part is stimulated, the whole labyrinth is stimulated. Hence, it is more appropriate to speak of "predominance of excitation" of one or another segment, according to the means used.

DENNIS, San Diego, Calif.

CLINICAL EXAMINATION OF THE OTOLITHIC APPARATUS. A. MOULONGUET, Oto-rhino-laryng. internat. **23**:610 (Dec.) 1939.

Otolithic and canalicular pathology are closely integrated, and it is needful to define their limits precisely. Effects produced by excitation of the otolithic apparatus may be classified into three groups: sensations informing the subject of the position of his head; reactional movements of the eyes, trunk and limbs; neurovegetative disturbances. The lapilli influence the tonus of the muscles that move the eyes, head, trunk and limbs in the sagittal plane. An increase of pressure of the lapilli on the maculas causes movement of the eyes upward and flexion of the neck, trunk and limbs; a diminution of pressure causes reverse movements. The sagittae operate in the frontal plane. An increase of pressure of the right sagitta causes rotation of the eyes to the right, an increase of tonus of the abductor muscles of the limbs on the right side and an increase of tonus of the adductor muscles on the left side, determining an inclination of the head and trunk to the left. Abnormal excitation of the otolithic organs produces vomiting, diarrhea, sweating and faintness. The pointing test is the most useful in the investigation of spontaneous signs. A normal person will point correctly in both the frontal and sagittal planes. One who has recently lost the function of the right labyrinth will point correctly in the sagittal plane but will past point to the right when tested in the frontal plane. If the head of a normal person is inclined to the left shoulder (which increases pressure on the right sagitta), he will still point correctly, by reason of an autoregulating mechanism. Pointing tests in the frontal and sagittal planes give precise information in regard to the functioning of the sagittae and the horizontal canals. If there is past pointing to the right of the right hand in a pointing test made in both the sagittal and frontal planes, a paralyzing lesion of the corresponding horizontal canal, not involving the sagitta, may be assumed. If the past pointing is to the right in the frontal plane but not

in the sagittal plane, it indicates a paralyzing lesion of the corresponding horizontal canal and of the sagitta. If there is no past pointing in the frontal plane, the integrity of the horizontal canal may be assumed; past pointing in the sagittal plane must be attributed to an otolithic lesion. Past pointing to the left, increased by bending the head to the right, indicates hyperexcitability of the right sagitta. Past pointing to the left, increased by bending the head to the left shoulder, suggests paresis of the right sagitta.

The lapilli are tested by pointing in the horizontal plane, moving the arm from the frontosagittal to the sagittal plane. If there is upward deviation, increased by bending the head forward, paresis of the lapillus corresponding to the arm being tested is indicated; if there is past pointing downward, increased by bending the head backward, hyperexcitability of the lapillus is assumed. Past pointing downward, influenced by change in the position of the head, is never the result of excitation of the semicircular canals.

Clinical methods for exciting the otolithic apparatus include the elevator test, the centrifugal test and the galvanic test. Practically, the galvanic and the pointing tests are sufficient. Binaural galvanism, with contrary poles, permits study of the excitability of the sagittae. The anode diminishes the sensibility of the nerve, and the cathode increases it. The semicircular canals are influenced by the current in the same way. In a normal person the results of the test are that there is no past pointing in the sagittal plane but there is past pointing in the frontal plane.

Binaural excitation with identical poles on the ears and the opposite pole on the chest permits study of the excitability of the lapilli. With positive poles on the tragi, upward past pointing (the pointing being done in the horizontal plane) occurs. The opposite effect occurs with the negative poles on the tragi.

Owing to the complexity of the problem, interpretation of the results of the tests is difficult, and only prudent conclusions, supported by repeated examinations, may be drawn.

A vestibular reflex from irritation, due to inflammation of the nasal sinuses, may pervert the functioning of the lapilli and provoke vertigo in the sagittal plane of diverse directions, according to whether the anterior or the posterior sinuses are involved.

DENNIS, San Diego, Calif.

SURGICAL TREATMENT OF ACOUSTIC TUMORS. SCHÖENBAUER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **46**:118, 1939.

Schöenbauer discusses the importance of a preoperative ventriculogram in all cases in which the presence of an acoustic tumor is suspected. The importance of this is stressed particularly in view of the fact that neither neurologic nor otologic studies can be of absolute diagnostic conclusiveness.

He first describes the technic of obtaining a ventriculogram, which usually serves as a guide to prognosis or to the relative size of the tumor. With the ventriculogram one can determine the degree of hydrocephalus and also the possibility of an occlusion in the region of the aqueduct. The author also describes his operative technic with the electrosurgical knife, which reduces hemorrhage to a minimum. He removes the bone over the occiput, exposes the cerebrum and, working forward, exposes the tumor. This he attempts to remove in toto with the capsule. Even though he is not always successful, he aims for a complete removal of the tumor mass.

His statistics show that in a series of 56 patients, he had 17.9 per cent immediate postoperative fatalities (6 patients with pneumonia, 7 with respiratory paralysis and 1 with meningitis). He was able to follow up carefully only 25 of the remaining 42 patients, of whom 9 were alive longer than three years, 9 were alive less than three years and 7 had died within the three year interval. The corresponding percentages are: three years and over, 28 per cent living; less than three years, 30 per cent living; postoperative deaths, 25 per cent, and unaccounted for, 16 per cent.

He discusses two common routes of approach to the acoustic tumor: the translabyrinthine and the posterior fossa route (his own). While he does not believe

that it is important whether a neurosurgeon or an otologist performs the operation, he feels that the taking of a ventriculogram is the most important preoperative procedure.

PERSKY, Philadelphia.

SURGICAL TREATMENT OF ACOUSTIC TUMORS. GÜTTICH, Ztschr. f. Hals-, Nasen- u. Ohrenh. **46**:122, 1939.

Güttich describes two methods of approach to acoustic tumors, the translabyrinthine (Panse) and the paralabyrinthine (von Seiffert). In the former technic, a complete radical mastoidectomy is performed first. The facial nerve is then exposed, and this acts both as a landmark and as a guide to the entire operation. The nerve is then followed to the geniculate ganglion and, in a direction parallel to the external auditory canal, to the posterior fossa and to the internal auditory meatus. The labyrinth is completely resected, and the dura of the cerebrum and the cerebellum is exposed. The superficial petrosal sinus is seen lying rather deep. After completing the resection of the labyrinth and so exposing the dura, the operator exposes the posterior surface of the petrous pyramid. To reach the cerebellopontile angle, he incises the dura from the sigmoid sinus to the internal auditory meatus. Since the facial nerve is completely exposed, it is often either injured or cut.

In the paralabyrinthine technic, the skull is opened below and posterior to the transverse sinus by a trephine opening. The opening is enlarged downward and backward toward the foramen magnum. The dura is then opened, and the median border of the cerebellum is exposed. The cerebral spinal fluid of the cisterna magna is drained by means of a paracentesis trocar. Then the lateral edge of the cerebellum is carefully elevated from the lateral surface of the petrous pyramid to the internal auditory meatus. The lateral cistern is drained, which exposes the lateral border of the cerebellum, the petrosa and the cerebellopontile angle to easy inspection. The tumor can thus be approached, and after enucleation of the tumor the dura is sutured and gauze drains are inserted into the superficial wound for the first few days.

The advantages of the labyrinthine method are that the dura is opened only toward the end of the operation and that there is less chance of herniation of the brain. However, there is a disadvantage in that the operative field is rather constricted, and when the tumor is large, difficulty may be encountered in its complete enucleation.

In conclusion, Güttich stresses the importance of early diagnosis, so as to anticipate extension and encroachment on the regional brain tissue. This is possible by a careful study of the history, a determination of the extent of involvement of the vestibular and cochlear nerves and evaluation of the roentgen evidence of widening or enlargement of the internal auditory meatus. He has performed 11 translabyrinthine and 2 paralabyrinthine operations and has had 3 immediate postoperative fatalities.

PERSKY, Philadelphia.

TREATMENT OF MÉNIÈRE'S DISEASE AND OTHER CONDITIONS ACCOMPANIED WITH TINNITUS AND VERTIGO BY VITAMINS. PAUL MØRCH, Acta oto-laryng. **27**: 629, 1939.

Mørch presents the case histories of 4 patients with symptoms typical of Ménière's disease, who also had symptoms of hypovitaminosis A and C.

The symptoms of hypovitaminosis A noted were: (1) conjunctival irritation and secretion, as well as more or less pronounced photophobia; (2) dryness of the pharynx and nasopharynx and at times irritation in the larynx; (3) loss of appetite and painful respiration; (4) loss of the sense of smell; (5) periodic attacks of thirst; (6) lassitude and listlessness; (7) necessity for long periods of sleep; (8) lumbago and myalgia.

The symptoms of avitaminosis C noted were (1) gingivitis and bleeding of the gums and irritation and redness of the buccal mucosa and the pharynx, (2) suggillations of the legs, (3) papillar eruptions between the fingers, (4) intense lassitude and (5) dyspepsia.

Mørch gave his 4 patients intensive treatment with vitamin A or C, as the case required, by intramuscular injection up to the point of producing hypervitaminosis. There was amelioration of the vertigo and tinnitus with improvement in the hearing of all 4 patients, but Mørch states that it is still too soon to determine the permanency of the results.

GROVE, Milwaukee.

Pharynx

SULFANILAMIDE IN SORE THROAT. PAUL S. RHOADS and M. L. AFREMOW, J. A. M. A. **114**:942 (March 16) 1940.

The authors present their conclusions from a two year study of 67 cases of sore throat, with detailed reports of clinical and laboratory data. Thirty-one patients received sulfanilamide in addition to the regular treatments; the remaining 36 patients, as controls, received every type of treatment but that with sulfanilamide. The subjects were all nurses of the Cook County Hospital, Chicago, and the Evanston Hospital, Evanston, Ill., who contracted hemolytic streptococcus pharyngitis or tonsillitis while on duty. It is apparent from the reports that the environmental conditions of the 67 subjects were similar.

The ultimate findings and deductions are as follows: 1. Sulfanilamide did not reduce the severity of the symptoms. 2. It did not shorten the period of incapacity. 3. It did not alter the incidence of complications. 4. It did not reduce the duration of the carrier state. 5. Toxic manifestations of other than the usual cyanosis occurred in one half of the patients who were given sulfanilamide.

The authors recognize the value of sulfanilamide, especially its proved merit in the treatment of severe Streptococcus haemolyticus infections of the deep structures of the throat, and do not wish to imply that sweeping generalizations are to be made from one series of controlled cases, but they question the routine use of this drug in the treatment of patients with uncomplicated tonsillitis or pharyngitis due to this streptococcus. They believe that no physician should be censured for withholding the drug in such cases unless complications, such as severe cervical adenitis, paranasal sinusitis, otitis media, mastoiditis or meningitis, supervene.

GORDON, Philadelphia.

ROENTGEN TREATMENT OF CHRONIC TONSILLITIS. VOGEL, Ztschr. f. Hals-, Nasen- u. Ohrenh. **46**:195, 1939.

Vogel reports a series of cases in which chronic tonsillitis was treated with high voltage roentgen radiation. Since the results were so varied and inconclusive, he believes that surgical removal of the tonsils should remain the procedure of choice, as this operation is usually safe and the results are rarely in doubt.

On the contrary, roentgen therapy produces rather uncertain results in that there is no indication that the tonsils can be completely resorbed or will become fibrotic or that the infection within the tonsils can be completely destroyed, and, finally, the treatment is always prolonged, requiring about three months for a complete course.

However, the author believes that this method of treatment is indicated for certain types of patients. These are the senile, arteriosclerotic, those with cardiac insufficiency and those with hemophilia and other allied conditions.

PERSKY, Philadelphia.

Larynx

BENIGN MILIARY LUPOID (BOECK'S TUMOR OF THE UPPER AIR PASSAGES). SEIFERTH, Ztschr. f. Hals-, Nasen- u. Ohrenh. **46**:69, 1939.

Seiferth describes a rare skin disease known as Boeck's sarcoid, or benign miliary lupoid, which presents local manifestations in the upper respiratory tract. His patient had an involvement of the skin of the face plus a fibrous swelling of the larynx in the region of the epiglottis. This had a thin pedicle and obstructed

the glottis to such an extent that the posterior commissure was not visible. No subjective symptoms were noted. The patient also showed an involvement of the lungs, which is characteristic of all patients with Boeck's sarcoid.

While local treatment to the larynx was not instituted, high voltage roentgen therapy to the skin, with a general regimen of antituberculosis therapy, was instituted. The laryngeal growth receded, and all evidence of the disease disappeared completely.

PERSKY, Philadelphia.

ROENTGEN EXAMINATION OF THE TUBERCULOUS LARYNX. W. MORITZ, Ztschr. f. Hals-, Nasen- u. Ohrenh. **46**:92, 1939.

Moritz classifies laryngeal tuberculosis as either sputogenous (bronchogenous) or hematogenous. He believes that he can differentiate these two forms, roentgenologically, since they present various idiopathic characteristics. In a transverse view of the larynx the sputogenous form is first shown by an elimination of the spindle-shaped shadow of the ventricle, due to thickening of the posterior laryngeal wall and swelling in the region of the arytenoids. As the disease progresses, this swelling extends from the arytenoids to the aryepiglottic folds and thus completely eliminates the shadow of the ventricle. In the hematogenous form the picture is somewhat different, since the disease process begins extralaryngeally and extends into the larynx. In the early stages there is a swelling of the epiglottis and the aryepiglottic folds and then of the arytenoid region, with subsequent swelling extending into the larynx, and finally there is thickening of the ventricular folds. As the disease progresses, necrosis and ulceration occur, with proliferations and granulations.

However, at certain times there may be difficulty in interpreting the nature of the disease process whether studied in the roentgenogram or in the laryngeal mirror. Consequently, Moritz uses a modification of the tomography technic which was advocated by Grossman and Chaoul. This is an anteroposterior examination of the larynx. The x-ray tube swings in a direction opposite to that of the plate, and in this way there is an accentuation of the area to be photographed, with an elimination or washing out of the structures surrounding the particular area. By this technic, he demonstrated various lesions in the larynx, and he believes that the technic is valuable for the following reasons: 1. It is possible to visualize lesions in both the glottic and the superglottic areas. 2. One can determine scar tissue structures. 3. One can determine the cause of difficult cannulization. 4. Laryngeal tumors are clearly outlined and delimited. 5. It is possible to follow up the results of therapeutic treatment. 6. It is an aid in the study of the physiology of voice production. 7. It is possible to differentiate easily between disease processes and foreign bodies in the larynx.

PERSKY, Philadelphia.

Nose

PRIMARY MELANOBLASTOMA OF THE NASAL CAVITY. L. NICOLAI and G. SAIBENE, Arch. ital. di otol. **51**:573 (Dec.) 1939.

A primary melanoblastoma of the nasal cavity is reported. The patient was a woman aged 74, and the clinical course of the disease extended over six years, death occurring from meningitis after a second operation. The result of a complete histologic examination of the tissues of the nasal cavity and of the specimen removed at the first operation is recorded.

Accounts of 50 similar lesions have been preserved in the literature. Melanoblastoma has been regarded by some writers as of mesenchymal origin, and by others as arising from the endothelium of the lymphatics. Still others have maintained that it is due to proliferation of the endothelium and the perithelium of the blood vessels. Nicolai and Saibene expressed the opinion that such a tumor developing in the nasal cavity may be assumed to have the same histogenesis as similar tumors in other parts of the organism. The conviction is spreading that the mesenchymal and nerve tissues are important points of origin of melanoblastoma.

DENNIS, San Diego, Calif.

DIFFERENTIAL DIAGNOSIS OF LUPUS VULGARIS (PERIARTERITIS NODOSA). RIECKE, Ztschr. f. Hals-, Nasen- u. Ohrenh. **46**:67, 1939.

Riecke describes the symptoms and differential diagnosis of periarteritis nodosa of the upper air passages. The onset is evidenced by severe rhinitis, which becomes purulent, with crust formation. The usual course is necrosis and destruction of the cartilage and the bony septum, with extension to the lateral nasal wall and sinuses. There follows formation of a saddle nose with destruction of the nasosinal attachment, injury to the optic nerve, protrusion of the bulb and blindness. Subjectively there are headache, frequent epistaxis, pain in the teeth of the upper jaw, rhinitis, obstructed breathing and a foul nasal discharge. In the external skin, pustules, small furuncles and abscesses developed, which are slow and protracted in their course. Further extension of the process may reach to the oropharynx, larynx and trachea.

These growths are difficult to diagnose by means of biopsy. There may be many miliary nodules, suggestive of miliary tuberculosis. However, there usually is a round cell infiltration about the blood vessels, with a granulomatous formation, which is rather characteristic of periarteritis. However, in the presence of irritation cells, especially those of the Langhans type, caseation may occur, so that the growth will again strongly resemble a tuberculous nodule. Other areas of periarteritis nodosa may be evidenced.

The author believes that the condition results from an allergic hyperergic tissue reaction to a generalized infection.

PERSKY, Philadelphia.

CHOLESTEATOMA OF THE MAXILLARY SINUSES. B. KECHT, Ztschr. f. Hals-, Nasen- u. Ohrenh. **46**:184, 1939.

Kecht reports a case of cholesteatomatous involvement of the left maxillary sinus, in which the tumor eroded through the orbital plate and presented a considerable amount of orbital swelling.

He performed a Caldwell-Luc operation and was able to eradicate the cholesteatomatous tissue, and the patient made a complete recovery.

The author suggests that the neoplasm may have been due to an injury to the face in early childhood.

PERSKY, Philadelphia.

THE NASOPHARYNGOSCOPE AS AN AID IN DIAGNOSING INFLAMMATORY CONDITIONS AND GROWTHS IN THE NOSE AND NASOPHARYNX. ZÖLLNER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **46**:214, 1939.

Zöllner describes the nasopharyngoscope and cites its value as an aid in diagnosis. With this instrument he was able to differentiate inflammatory conditions of the orifice of the eustachian tube and growths (adenoid, lymphoid, malignant) in the postnasal region and the ethmosphenoid recess and about the eustachian orifice. He also diagnosed tuberculous processes in this region, and he believes that a rhinoscopic examination is incomplete if one does not employ this instrument as a routine procedure. Without its use many important pathologic conditions are completely missed.

PERSKY, Philadelphia.

Miscellaneous

SYNDROME OF AVELLIS, ASSOCIATED WITH HEMIPARESIS OF THE NINTH AND TWELFTH NERVES AND WITH THERMOANALGESIA OF SYRINGOMYELIC TYPE. V. BORGIOLI, Arch. ital. di otol. **51**:594 (Dec.) 1939.

Borgioli reports the case of a man aged 80 who was suddenly stricken with paralysis of deglutition. The attack was not accompanied by loss of consciousness or by vertigo. The Wassermann test was negative. Examination revealed paralysis of the pharynx, palate and larynx, gustatory anesthesia of the posterior part of the tongue and hypokinesis of the tongue, all on the right side, and thermoanalgesia involving the left limbs and the left half of the trunk. The condition

was much improved after a stay of six weeks in the hospital. The pathologic condition was assumed to be hemorrhage in the field supplied by the postero-inferior cerebellar artery, affecting particularly the crossed sensory fibers in the reticulated gray substance of the bulb. DENNIS, San Diego, Calif.

HISTOLOGIC CHANGES IN THE ESOPHAGUS, RESULTING FROM THE INGESTION OF CAUSTICS. V. SANGIOVANNI, Arch. ital. di otol. **52**:1 (Jan.) 1940.

After applying potassium hydroxide, phenol, mercury bichloride, silver nitrate and sulfuric acid with a swab to the lining of the esophagus in 14 dogs, Sangiovanni studied the effect on the animals of these cauterizing agents. Most of the dogs died from the lesions within a few days; 1 was kept alive for four months by gastrostomy, and another, with severe lesions of the esophagus and without gastrostomy, lived for five months; 3, with insignificant lesions, were killed three months after the tests. Autopsy and microscopic examination of the tissues were carried out in all cases. The gravest lesions encountered were those from mercury bichloride. These occurred not so often in the esophagus as in the kidneys and killed the dogs in from ten hours to three days; in all cases an intense edema of the cauterized regions was found, and noteworthy were the deposits of calcium salts in the renal tissues, due to the decalcifying action of the mercury on the bones.

The anatomic lesions encountered soon after the cauterization varied in intensity with the caustic employed. Sometimes only the mucosa was involved, but usually both the mucosa and the submucosa were compromised. Phenol produced less profound local lesions; grave destructive lesions, leading even to the destruction of neighboring organs, were produced by potassium hydroxide; the lesions produced by silver nitrate were superficial, but interesting is the observation of deposits of silver in the renal tissues; the animal which was submitted to the action of sulfuric acid (50 per cent solution) evinced no symptoms, although the cauterization was repeated after one month. This dog was killed after three months, and examination revealed no lesions except a slight thinning of the epithelial layer.

The zones showing the more intense lesions were those corresponding to physiologic constrictions. Of the 4 dogs on which phenol was used, death occurred in the first in three days and was caused by toxic nephritis and bronchopneumonia; the second also died in three days, from a general toxic state, hemorrhagic nephritis and bronchopneumonia; the third dog lived seventeen days, and the fourth was killed after four months, and in both reparative phenomena and consecutive stenosis had followed the destructive process. The evolution of the lesions from potassium hydroxide was very severe by reason of the profound alterations characteristic of the action of this substance; one dog died in six days with tracheoesophageal fistula, the second in twenty-five days, from inanition because of a cicatricial stenosis of the esophagus, and the third died after five months from inanition due to cicatricial stenosis of the esophagus and periesophagitis. To be noted also in the survivors was the appearance of dilatation of the esophagus above the stricture.

The results of the experiments agree with the observations made on human beings subjected to the ingestion of caustics, except that in the dog cauterized with sulfuric acid no grave lesions were found. This fact may be dependent on local factors or the use of an unsuited concentration. The stenoses were analogous to those observed in man as to their location, their histologic aspect and the occurrence of dilatation above the strictures. Noteworthy was the occurrence of mid-gastric stenosis due to perigastritis in a dog which was cauterized with phenol. Sometimes caustics may produce slight lesions of the esophagus and much graver lesions in the stomach.

Detailed protocols of the cases are furnished. DENNIS, San Diego, Calif.

MERCURIAL DISINFECTION EMPLOYED IN LOCAL TREATMENT IN OTORHINOLARYNGOLOGY. M. BAER, Monatschr. f. Ohrenh. **73**:751 (Dec.) 1939.

Merfen, a new mercurial disinfectant, has been investigated from all angles, and its advantages over the usually employed agents are given. In rhinologic practice it is applied best in combination with glycerin. The author advises the use of merfen as an active and nonpoisonous means of disinfection.

LEDERER, Chicago.

RESULTS OF FOURTEEN YEARS' TREATMENT OF LUPUS OF THE MUCOUS MEMBRANE BY RADIUM. E. KNAPP and H. LOEBELL, Ztschr. f. Hals-, Nasen- u. Ohrenh. **46**:73, 1939.

The authors describe the technic of applying radium to lupus at various sites in the nose and throat. They also describe the use of suitable adapters, applicators and prostheses, since these are important in the proper placing of the radium.

They analyzed 305 cases of lupus, in 280 of which the mucous membrane of the anterior portions of the nose was involved, in 98 that of the mouth, in 26 that of the pharynx, in 49 that of the larynx and in 7 that of the lacrimal ducts. There was associated involvement of the external skin of the face in 243 cases, of that of the buttock in 21 and of that of the extremities in 31. In 55, or 16.8 per cent, the lesions were localized to the mucous membrane alone, and in 33, or 10.7 per cent, there was associated pulmonary tuberculosis. In 4 cases the lesions were associated with syphilis and in 8 with carcinoma.

In 253 of the 305 cases, treatment was either with radium alone or by irradiation in combination with some other therapeutic measure. The authors report cures in 196 cases, improvement in 28, questionable results in 54, failures in 27 and deaths in 8. Of the 196 patients cured, 164 were treated with radium alone.

The authors state that, while there have been reports of a great number of spontaneous cures of lupus of the mucous membrane, particularly when this involvement was associated with lesions of the skin, their feeling is that radium is an important adjunct to treatment, and they are not in agreement with the hopeless prognosis that is made in some clinics.

PERSKY, Philadelphia.

TREATMENT OF LUPOID CARCINOMA. ZANGE, Ztschr. f. Hals-, Nasen- u. Ohrenh. **46**:84, 1939.

Stressing the fact that in many cases lupus undergoes malignant changes, the author expresses the belief that it is important to remove the growth. He advocates electrosurgical removal. He describes in detail a number of cases in which such treatment was used and in which there was no recurrence after three and one-half years.

PERSKY, Philadelphia.

MENINGIOMA OF THE RIGHT MIDDLE FOSSA INVADING THE TEMPORAL BONE. BEHREND and BARTH, Ztschr. f. Hals-, Nasen- u. Ohrenh. **46**:145, 1939.

Behrend and Barth report an interesting case of meningioma. The growth was removed in 1927. The clinical picture at that time showed a series of symptoms lasting from one to two years. These were principally tinnitus, deafness and facial weakness. At the time of operation the tumor was found to originate from the dura and seemed to be adherent to the temporal bone. However, in the subsequent course there seemed to be a recurrence of this growth, which infiltrated the entire temporal bone.

In 1938 the patient died of septicemia and sinus thrombosis due to an underlying cholesteatoma. This cholesteatoma apparently did not come from an old tympanic perforation but seemed to represent rather a metaplasia occurring in the posterior superior canal or in the meningioma.

The authors presented photographs showing the original tumor, the extensive infiltration into the temporal bone, the marked cholesteatomatous mass in the mastoid process and the generalized infiltration of the entire temporal bone proper by this tumor. In addition, they made an extensive survey of the literature.

PERSKY, Philadelphia.

NASAL AND ORBITAL COMPLICATIONS OF SCARLET FEVER. STEFAN KREPUSKA, Ztschr. f. Hals-, Nasen- u. Ohrenh. **46**:175, 1939.

In the two year period of 1937-1938, a study of 797 cases of scarlet fever revealed severe sinal complications in 61. In 28 of these, the sinal condition was associated with some orbital involvement. The maxillary and the ethmoid sinuses were the most frequently involved. Since the skeletal arrangement of the nose and face at many sites is composed of cartilage, connective tissue and mucous membrane, an infection can readily involve these areas, and since the suture lines are not completely closed, the infection can readily spread from the sinus to the orbit.

The author advises conservative treatment, instituted early. This should comprise shrinkage, suction, irrigation, fracture of the middle turbinate and antral puncture. However, when these conservative measures fail to produce a subsidence of symptoms within twenty-four to forty-eight hours, radical surgical intervention should be considered. Depending on the amount of involvement, the following procedures are indicated: the Caldwell-Luc operation, ethmoidectomy or the external frontal sinus operation (Killian). He believes that intranasal surgical operations are less effective than the more radical procedures. In the series of 61 cases, he reported only 1 death following any of these procedures.

PERSKY, Philadelphia.

Society Transactions

MONTREAL MEDICO-CHIRURGICAL SOCIETY, SECTION OF OTOLARYNGOLOGY

D. H. Ballon, M.D., *President*

E. A. Stuart, M.D., *Secretary and Abstract Editor*

Oct. 17, 1940

Giant Cell Tumor of the Hard Palate; Parathyroid Adenoma; Hyperparathyroidism. Dr. D. H. Ballon.

A woman aged 22 years was admitted to the hospital on May 28, 1940. Four months previously a mass appeared on the roof of her mouth. It increased progressively in size and was painful to touch. When the mass first appeared, the patient was ill with fever, general malaise and loss of appetite. One and a half years before admission she noticed a firm painless tumor near the right lobe of the thyroid, which grew steadily; at the time of admission it was the size of a pigeon's egg. There was nasal intonation. The patient was poorly developed and pale and appeared ill.

Examination showed a smooth oval almost cystic swelling, about 5 cm. long and 2 cm. wide, localized to the hard palate. There were a few palpable posterior cervical glands.

The Wassermann reaction of the blood was negative. A biopsy specimen of the tissue was found to be very soft, friable and vascular. The pathologic report was "giant cell tumor." A parathyroid adenoma was removed by Dr. Baxter. The hemogram indicated a deficiency in bone marrow response, probably due to an infectious or toxic process of long standing. The red blood cells numbered 3,500,000 and the white blood cells 3,900 per cubic millimeter; the hemoglobin content was 64.5 per cent; the platelets numbered 198,000. Bleeding time was not appreciably delayed. Coagulation required five and four-tenths minutes; with calcium, nine minutes. The prothrombin time was normal. The blood calcium was markedly increased (14.45 mg. per hundred cubic centimeters).

DISCUSSION

Dr. W. DeM. Scriver: This patient was first seen in the outpatient department about the beginning of 1940, with a tumor of the palate. She was next admitted to the gynecologic clinic for an abortion. In the outpatient department it had been noted that she had a tumor of the metatarsal bone and also general bone decalcification. These multiple tumors were reason for suspecting that there was something wrong with her metabolism of calcium. Solitary or multiple cysts of bone result from oversecretion of parathyroid hormone. Patients with this abnormality have been called "calcium diabetics." This patient's blood phosphorus was diminished. A chart was presented to show the normal blood calcium level at 10 mg. per hundred cubic centimeters, while this patient's was 16 mg., and normal blood phosphorus at 3 mg., while this patient's was 1 mg. She was excreting in the urine more calcium than she was taking in her diet, and in the stool she was excreting as much calcium as she was taking in her diet. It was decided that this was a case of hyperparathyroidism. The patient had a parathyriod tumor, which was removed and was identified by histologic section. Two weeks after operation tetany developed. A month was required to bring this under control. Since her operation she has suffered a pathologic fracture of the femur.

In the roentgenograms taken before operation the femur showed no fracture. Since operation her blood calcium and blood phosphorus have returned to normal levels and she has become otherwise clinically and chemically normal. The diagnosis of parathyroid tumor depends on the presence of multiple cysts and decalcification of bone. If one can feel the tumor as well, the diagnosis is more certain to be correct.

DR. STEWART BAXTER: I had seen this woman in the outdoor department when she first came complaining of the tumor of the palate and swelling of the dorsum of the foot and of the neck. I recommended her for admission to the hospital, but she failed to comply with my request and was next seen in the department of gynecology. Tumor of the parathyroid is of great interest. It was first described in 1891, by von Recklinghausen. At that time little was known about the pathologic factors. About 1904 an autopsy was done in a case of osteitis fibrosa cystica. Later, the fact was mentioned that there might be some connection between the parathyroids and this condition of the bones. When extracts containing the active factor of the parathyroids became available for experiment, a definite correlation between parathyroid tumor and the syndrome of osteitis fibrosa cystica was suspected. Since then over 200 cases of this syndrome have been proved to be cases of hyperparathyroidism. Parathyroid tumors are divided into the following groups:

1. Simple hyperplasia.

2. Adenoma, usually of one gland. These frequently are cystic and may reach considerable size. The tumor in this patient was an adenoma about the size of a walnut with the shell on and was causing displacement of the right lobe of the thyroid, with a resultant mass in the neck.

3. Malignant tumors, which are rare.

As a rule, if the tumor can be found, the treatment is surgical. This is not always as easy as it seems. In many cases the neck has been explored several times before the adenoma has been found. Usually then it has been found in the mediastinum. After removal of the whole tumor an acute phase of tetany is apt to occur, which may be difficult to control. If the disease is severe, with high blood calcium and destruction of bone, only partial removal is advised, a piece being allowed to remain until normal activity is regained. In certain cases only one parathyroid is present, the removal of which would lead to serious chronic tetany.

The operation on this woman was interesting. The right lobe of the thyroid was enlarged, and there was a cystic swelling, which it was almost impossible to separate from the gland. The cyst contained a bright yellow fluid. Usually a thyroid cyst contains brownish bloody fluid. Hoping to prove which part of the gland was thyroid and which was parathyroid, we had a pathologist make some sections. Unfortunately, all the sections came back with the report "normal thyroid." I explored both sides of the neck and the anterior mediastinum without seeing anything that looked like a parathyroid tumor. I took out part of the thyroid and the cyst. A short time afterward further section of the tissue removed was reported as parathyroid, so that the whole cyst, which lay behind, was a parathyroid tumor which had become cystic, causing the symptoms. It took considerable time for the patient's blood calcium to become normal, but it is now perfectly normal.

Two Cases of Otitic Meningitis Due to Streptococcus Haemolyticus Beta.

DR. KEITH HUTCHISON.

CASE 1.—A woman aged 30 years was admitted to the Montreal Neurological Institute on May 6, 1940. She had been suffering for three days with neuralgic pains in the head, slight head cold, stiffness of the neck, drowsiness and difficulty with speech. For forty-eight hours she had had a blood-stained discharge from the left ear. On examination her temperature was 101 F.; she was semiconscious, and there was some stiffness of the neck; the retinal veins were dilated, and there

was depression of all the reflexes. The left tympanic membrane was perforated, and there was a watery bloody serous discharge in the aural canal. The throat contained stringy mucus and appeared injected.

On admission the spinal fluid pressure was 480 mm. of water; the fluid was opalescent; the Pandy reaction was 3 plus, and there were 1,450 cells. A culture of the cerebrospinal fluid showed a light growth of Streptococcus haemolyticus. Cultures of material from the ear showed a heavy growth of Streptococcus haemolyticus beta. Roentgenograms showed minimal changes in the left mastoid.

On May 6 a paracentesis of the left tympanic membrane was carried out, to improve the drainage. On May 7 a simple mastoidectomy was performed. At the time of operation the patient's condition was grave. She was practically comatose. The operation disclosed an extensive pneumatization of the mastoid. Pus was present throughout. There was no breaking down of the cells. The dura was not exposed, and the sinus plate was intact. The lateral sinus was exposed and appeared normal. At the end of the operation the patient's condition was desperate. The pulse was 178. Cutting down on the vessels at the elbow in an effort to administer dextrose intravenously revealed that no blood was present in fairly large veins.

From admission the patient was treated with sulfapyridine sodium (the monohydrate sodium salt of 2-[paraaminobenzenesulfonamido]-pyridine), and her condition gradually improved. On the day of the mastoidectomy her temperature was 105 F. On May 11 administration of sulfapyridine sodium was discontinued because of toxic manifestations revealed by a drop in the white blood cell count to 5,600 per cubic millimeter. The temperature returned immediately to 105 F. On May 12 a culture of the cerebrospinal fluid yielded no growth. On May 13 some clouding of the right mastoid was noted roentgenologically. Administration of the drug was resumed and was accompanied with remission of the temperature to 101 F. On May 16 administration of sulfapyridine sodium was discontinued, and on May 18 sulfanilamide (15 grains [0.97 Gm.] every four hours) was given by mouth. This was continued until May 20, when sulfapyridine sodium was again administered for a period of seventy-two hours. No further chemotherapy was administered to this patient.

On May 25 otitis media developed in the right ear, and spontaneous rupture occurred two days later. Routine treatment was instituted, but the ear continued to discharge profusely, so that a mastoid operation was carried out on June 10. A picture similar to that encountered on the left side was present. From this time on the patient made a slow but steady recovery. During the latter part of her stay in the hospital she experienced some epileptiform attacks, which were relieved with phenobarbital.

The highest level of sulfapyridine in the cerebrospinal fluid was 8.6 milligrams per hundred cubic centimeters.

Case 2.—A girl aged 9 years was admitted to the Montreal Neurological Institute on July 12, with a history of an attack of measles early in June, associated with acute suppurative otitis media on the right and followed by a simple mastoidectomy on June 27. The patient was sent to the Montreal Neurological Institute on account of periodic pain in the right side of the head since operation and some neurologic signs suggestive of brain abscess. On July 13 a lumbar puncture showed a spinal fluid pressure of 140 mm. of water; 5 cc. of spinal fluid was removed and appeared clear; 25 lymphocytes were present, with some red blood cells, which were considered traumatic. The Pandy reaction was negative. It was evident that residual infection was present in the right mastoid cavity. There was a purulent discharge from the meatus, while a fistula at the most dependent portion of the mastoid wound was pouring pus. The incision had been sutured and had healed securely. On July 14 a revision of the simple mastoidectomy was carried out. The mastoid had been exenterated fairly well. There was a large exposure of dura just over the aditus. The aditus had not been thoroughly opened. Posteriorly in the superior portion some bone was

removed, which contained infected mastoid cells. One of these was in open contact with the dura. In the original operation the dura had been exposed also at a second point, over the tegmen behind the aditus. The lateral sinus was exposed and appeared healthy. The wound was packed with dry iodoform gauze and left open.

On July 15 the child's condition became more serious. The temperature was 106 F., and she was drowsy. Dr. A. R. Elvidge performed a lumbar puncture, and the spinal fluid appeared milky, with a large number of cells. The patient was treated actively with sulfapyridine sodium in large doses. On July 17 a partial anuria occurred and it was necessary to withdraw the drug for a short time, but administration was subsequently resumed.

The child made an excellent recovery and was discharged five weeks later with a healed wound, the middle ear dry and the hearing good.

DISCUSSION

Dr. A. R. Elvidge: It is always interesting to follow the cases of patients who are doing well, and these 2 patients did remarkably well. I saw both of them while they were being treated. Reading the summaries of these two histories, one notices how rapid was the onset of the meningitis in each case—developing within one or two days of the onset of pain in the ear or aural infection. The patient in the second case, I remember, came in supposedly with a cerebellar abscess. As she had few signs to support that diagnosis, a lumbar puncture was done, and the pressure of the spinal fluid was found to be normal. The mastoidectomy, which was done outside, was not complete. Many infected cells were still present, so Dr. Hutchison undertook to complete the mastoid operation. After about twenty-four to forty-eight hours all the signs and symptoms of meningitis suddenly developed, and the spinal fluid pressure then was much elevated. The patient was treated for meningitis. Often after head injuries one sees an acute onset of meningitis. A patient may have a fracture of the skull, e. g., in the anterior fossa, and within twenty-four to forty-eight hours all the signs and symptoms of meningitis may develop suddenly. There is always the question then of differential diagnosis. At operation it is a question whether one should not determine what the dura looks like in the middle fossa and also over the lateral sinus. This information, if a complication should arise, would give some clue as to whether the posterior or the middle fossa was involved. The interesting thing now is the method of treatment which in the last year or two has become so extremely successful in cases such as these, which before were so likely to terminate fatally. At the Montreal Neurological Institute we use sulfanilamide or sulfapyridine, but we are still forcing fluids freely in such cases and doing frequent lumbar punctures. In the children's hospitals, I think, physicians are not stressing the importance of forcing fluids and are getting good results. I have seen 2 or 3 patients in whose cases fluids have not been forced and lumbar punctures have not been made who have not done so well. The reason for frequent lumbar puncture is partly that this procedure has been attended with success in the past. The administration of plenty of fluid enables the patient to have frequent washing out of spinal fluid. When these drugs are being used, they should not become too concentrated in the blood, and the kidneys must be allowed to excrete them freely. I remember a patient at the Montreal General Hospital who was treated by chemotherapy and, from a scientific point of view, recovered from meningitis. She had not had large amounts of fluid, and toward the end of convalescence pachymeningitis and a subdural abscess developed as complications. Whether this was due to the fact that she had a low intake of fluids, I don't know. I know of a boy in whom hemiplegia developed during the course of treatment, at a time when his fluid intake had not been kept up. Dr. Hutchison's first patient had an interesting complication. Hemiparesis and convulsions developed, and the question arose as to whether she had an abscess of the brain. That possibility was finally eliminated by lumbar

puncture, and it was concluded that her symptoms must have been due to a cerebrocortical thrombosis. So far as streptococcic meningitis is concerned, I can remember 2 or 3 patients who recovered without recourse to chemotherapy. One at the Montreal Children's Hospital recovered from an extremely acute streptococcic meningitis after repeated lumbar punctures, forcing of fluids and injection of small amounts of polyvalent serum (mixed streptococcus strains of the scarlet fever type). I remember another patient at the Montreal General Hospital who recovered after treatment with serum and the forcing of fluids. The great majority of cases of streptococcic meningitis previously terminated fatally. I think the greatest triumph of all is in the treatment of pneumococcic meningitis, a disease from which there was practically no hope of recovery until the introduction of sulfapyridine. The pus always became so thick that eventually the spinal fluid could not be drawn off and hydrocephalus resulted. In streptococcic meningitis the fluid remains much more dilute. It might become opalescent but not turgid as in pneumococcic meningitis. After treatment with sulfapyridine and the forcing of fluids and drainage, the majority of patients seem to get well without residual symptoms, in spite of the fact that they have been so ill. I think in the last year or so I have seen 6 patients with pneumococcic meningitis recover and have observed no deaths from the infection. The disease runs a stormy course, especially in traumatic cases, as the organisms tend to reinfect from the area of trauma. In these cases I have combined chemotherapy with the use of serum. All manner of complications occur, but the patients have eventually gotten better. Sometimes the chemotherapy has to be discontinued, or the administration of serum is discontinued and is continued later.

Dr. W. P. E. Paterson: Recently I had a case of meningitis following mastoiditis. I put the patient on a regimen of sodium sulfapyridine and serum. When I took her to the hospital she was unconscious, and about twenty-four hours later she became conscious. Her temperature dropped from 105 F. to 99 F., and she seemed to get along fairly well for a couple of days. The administration of serum was then discontinued, and that of sulfapyridine sodium was continued. She became unconscious. I again gave her serum, and she was better. I then reduced the dosage of sulfapyridine sodium, and she became unconscious. Chemotherapy and administration of serum were continued for several days. After a stormy course the patient began to improve. After two weeks of gradual reduction in therapy she was thought to be all right, when suddenly she died. The second day she was in the hospital, I attended a meeting at which chemotherapy was discussed. It was said that the concentration of sulfapyridine in the blood should not be higher than 10 milligrams per hundred cubic centimeters. When I made a determination, the concentration in my patient's blood was 25 milligrams.

Dr. Keith Hutchison: You have heard the cases reviewed by Dr. Elvidge, and I think there is not much I can add. At the Royal Victoria Hospital in the past few months several cases of otitic meningitis have been observed. In 1935 we had 6 patients with the disease, and they all died. In 1936 we had 6, and they all died. In 1937 we had 5, and 1 recovered—a youth who was treated with sulfanilamide. His was an interesting case. He was the patient Dr. Elvidge mentioned, in whom hemiplegia developed. He is now able to attend high school and is quite normal, but he had to be reeducated completely as to eating habits, etc. In 1938 we had 5 patients with the disease. The first 3 died and the others recovered. By this time we were using chemotherapy. In 1939 all the patients recovered, and as to 1940 I think I can say the same thing, for this year 5 or 6 patients have recovered. We are thankful to have Dr. Elvidge and Dr. Wilder Penfield and their group of skilled associates to handle these cases. There is no doubt that the lumbar punctures every four hours or every six hours are a most important part of the therapy, while keeping the fluid level up with constant change. Severe infections have to be treated by intravenous administration of sulfapyridine sodium. Both of the cases presented are interesting, the

first because of the rapidity of the onset. The patient was finally taken to the operating room, and a mastoidectomy was done. At the end of the operation she was in very poor condition, her pulse rate being about 178. She was in such a condition that when a vessel was cut, there was no bleeding. However, she did well. The neurologist's only comment now is that she had a changed personality, but I imagine that any one who spent two months in the Neurological Institute would have a changed personality. The second case was interesting from an otolaryngologic standpoint. There are two schools of thought as to whether one should close a mastoidectomy wound or leave it wide open. We of the Royal Victoria Hospital, after our splendid training from Dr. H. S. Birkett, belong to the open drainage school. We do not suture the wound after an operation for an acute involvement of the mastoid. I don't think closing of such a wound saves much time. In the second case reported, the wound had been sewed up tight, and three weeks later we had to cut through the healed scar. There was a little fistula at the lower part of the wound, through which pus was draining. Pus was also draining from the middle ear. Some cells had not been cleared out, but a fairly good mastoidectomy had been done, and we otologists know that we do not often remove all the cells. A few areas had not been opened, but I think the infected cells in these areas might have broken down and drained out if the wound had been left open. The patient's temperature was 106 F., and her spinal fluid looked milky. With the use of chemotherapy well established, many more cures should be possible.

A Case of Facial Tic. DR. G. E. TREMBLE.

A woman aged 31 was first admitted to the Montreal Neurological Institute on Nov. 10, 1936, complaining of a twitching of the right side of the face, including the right lower eyelid, right upper eyelid, right cheek and right side of the mouth, of one year's duration. At that time there were a fine nystagmus on gaze to either side, more distinct to the left and not well sustained; voluntary and emotional weakness of the right side of the face; diminution of the gag reflex, and slight conjunctivitis on the left. Roentgenograms showed definite decalcification of the right petrous apex. Encephalography revealed no evidence of an expanding lesion or of a contracting cicatrix. The right seventh nerve was more reactive throughout to galvanic current than was the left. The results of hearing and vestibular tests were within normal limits. The tearing test showed a slight diminution of lacrimal secretion on the right side. After investigation the patient was discharged with a diagnosis of "facial tic, right (irritative lesion of the right seventh nerve)." In February 1937, at St. Luke's Hospital, she had the facial nerve stretched near the stylomastoid foramen. About eighteen months after this the twitching reappeared. It started in the right side of the face, with twitching of the right lower eyelid, and progressed so that there was continuous twitching of both eyelids, the right cheek and the right side of the mouth. She stated that it was sometimes quiet for twenty minutes at a time but always started as soon as she saw some one, especially a stranger. When she was out walking and when she was excited, it became more severe. There was also spasm of the face, lasting several minutes at a time. In November 1939 she reported that she was unable to swallow. It seemed as if something were pressing on her windpipe. This condition lasted for four days. She had great difficulty in swallowing, choking when she attempted it. On Nov. 28, 1939, roentgenograms revealed no demonstrable lesion of the esophagus. An electrogram showed no cortical discharge corresponding to a spasm of the right side of the face. In March 1940 the patient was again admitted to the Montreal Neurological Institute, with fulness of the retinal veins, blurring of the margins of the optic disks and slight filling in of the physiologic cups, facial paresis on the right side and exaggerated reflexes in the upper extremities. The impression was that she had a recurrent facial tic and facial paralysis on the right. Additional investigation during this period revealed no increase in either lacrimation

or salivation on the right side. Roentgenograms of the skull revealed no evidence of mastoiditis. Both ear drums were definitely retracted but normal in appearance. There was slight inflammation of both tonsils, especially on the left. She was admitted to the otolaryngologic service on May 14, and on May 15 a modified radical mastoidectomy, with exposure of the seventh nerve, was performed. The cells were normal. The bone over the vertical part of the facial nerve was removed, as well as part of the external auditory canal. The facial canal was opened from the stylomastoid foramen up to the first turn, a distance of about 12 mm., and along the turn transversely for about 4 mm. The facial canal was left open, and light packing gauze was inserted. Except for a small drain at the bottom the wound was sewed up. When the patient awoke from the anesthesia, the right side of the face was paralyzed except for a faint wrinkle on the right side of the forehead. The twitching has not recurred. The lower branches of the facial nerve are still affected, but it is hoped they also will improve. The movement is gradually improving, and the patient can close her right eye normally. The nerve appeared to be free, but a fine probe was passed along each side of the descending portion, to make sure that no adhesions were present, especially near the lower opening. Macroscopically the appearance of the nerve was normal, with no sign of swelling along its course.

DISCUSSION

DR. G. E. TREMBLE: Before Dr. Penfield discusses the case, I should like to mention a few points that were not brought out in the synopsis. In 1934 Duel reported several cases in which he had operated for long-standing Bell's palsy. At that time he said: "I believe that although operative treatment of severe Bell's palsy is not yet a fixed principle, yet with continued study of indications for early intervention, it will eventually become so."

The difficulty at present is that observers recognize only the severe forms of the disease, by the fact that the patients never recover. The otolaryngologist sees a patient a year or more after the onset of the illness, when deformity has developed, and he is deterred from advising operation at that stage by the feeling not only that a complete cure is unlikely but that an operation may jeopardize the slight recovery which has already occurred. One so often feels that if only one could have operated in the first few days after the palsy developed, one could have saved the patient a permanent deformity. There is ample evidence that early decompression will produce immediate recovery. At times the paralysis begins to disappear before the patient leaves the operating table. The case I have reported today is such a one. But when the patients are not seen in the first few days of their paralysis, it is often difficult to decide whether or not to recommend an operation, when it is known that 4 out of 5 patients with Bell's palsy recover spontaneously. The otolaryngologist, however, is interested in the remaining 20 per cent, and it is precisely in these cases that the evidence indicates a more central involvement, extending to the genu facialis. As already mentioned, the conception of herpes oticus implies such a central involvement. Furthermore, the phenomenon of tearing suggests a ganglionic involvement, too. It is clear that the syndrome could not occur unless the large superficial petrosal nerve had been involved, and that can occur only if the facial nerve is strangulated in its first part. Four years ago Dr. Penfield and I reported such a case.

Sanders has recorded a case in which he decompressed the facial nerve down to the ganglion. This involves exenterating the tympanic membrane, together with the malleus and incus—a serious addition to an already laborious and difficult procedure. Furthermore, the risk of labyrinthine infection cannot be ignored, especially in cases of herpes oticus, in which the vesicles are likely to be already secondarily infected. I do not believe that complete osseous decompression is ever likely to become a routine procedure. Tempea has recorded several cases in which he merely uncapped the fourth portion of the facial nerve (limited osseous decompression) and obtained dramatic cures.

The interesting question arises: How can this limited operation succeed if, as I have suggested, the site of strangulation is in the first part of the nerve? Consider the canal. Its contents—nerve, stylomastoid artery, vein, lymphatics, etc.—are believed to occupy from one third to one half of its capacity. When palsy is developing, swelling, it may be assumed, is taking place. Remember that the stylomastoid artery, in addition to nourishing the nerve, carries a large volume of blood for the supply of the tympanic membrane and the adjacent bone. In the presence of inflammation this vessel is probably enormously increased in size. The result is that the thin-walled veins and lymphatics are obliterated. A vicious circle ensues. Blood can get in but cannot drain out, and so the nerve dies. But suppose that the physician ablates the engorged bone to which the artery is carrying this great volume of blood. Presumably the artery will sink down to normal caliber, and so just enough room might be given for the vein to open and the nerve to reexpand. I suggest, then, that these osseous decompressions may be achieving results by virtue of the incidental vascular decompression. If this is correct, may it not be possible to abandon the laborious osseous decompression? Can vascular decompression be achieved by any simpler and less dangerous method?

Dr. Wilder Penfield: Dr. Tremble has discussed Bell's palsy, and I understand that my discussion is to be about tic of the seventh nerve. I first saw the girl whose case has been reported in 1936, when she was complaining of spasms of the right side of the face and twitching of the right lower eyelid, which were gradually increasing. She had some noise in the right ear, in addition. She was interested particularly because of the cosmetic effect. The twitching was almost continuous. At that time the physical examination revealed no other abnormality except some decrease in the function of the seventh nerve. There was no increase of tearing. Her hearing was not decreased. There was some rarefaction of the mastoid bone on the right side. The patient was observed and asked to come back. She went to St. Luke's Hospital and had the seventh nerve stretched, which gave her complete paralysis in the regions supplied by that nerve. She preferred the paralysis to the spasm. Function came back in the nerve, however, and she began to have the spasm again. When, after this, she came to us for reexamination, Dr. Tremble laid bare the nerve to see if he could find the cause of the irritation. There is no well established lesion to explain the condition. Harris, of London, felt that there was an irritation beyond the geniculate ganglion. It is somewhere along the course from the nerve cell out. If the absence of spasm after operation is continuous, this procedure must constitute the new approach to the condition. If this treatment does not work, the best thing to do is to cut the nerve outside and anastomose it to the twelfth This operation restores motion of the face to a moderate extent. It gives tone and movement, although both are imperfect. I had a patient with a spasmodic attack, which began with marked tearing. This indicated that her lesion was proximal to the genu. Various fibers can be involved in that discharge. It is a curious thing that the cranial nerves are involved in a lesion of this sort. In the fifth nerve there is tic douloureux; in the ninth, glossopharyngeal neuralgia, and within the eighth, Ménière's disease, which is a spasmodic discharge. The lesion of the seventh nerve observed in the case reported and a discharge in the motor division of the fifth nerve can also produce roaring in the ears, due, I suppose, to the innervation of the tensor tympani. I once encountered a case in which the muscles of mastication were also involved, and the patient complained more of the noise in the ear than of the spasm of the muscles of mastication. This seems to me the most constructive approach to the pathologic condition of one of these nerves.

Dr. E. A. Stuart: Will Dr. Penfield please tell us what the difference is between "tic" and "spasm"?

Dr. Wilder Penfield: "Tic" is a word that has been used for a long time and, I think, has come to mean any kind of sudden seizure. "Tic," properly used,

I think, means "spasm." A facial tic, properly speaking, is this which has just been described. Habit tics, or movements, are voluntary.

DR. E. A. STUART: Does a facial tic come on with voluntary movement, or is it involuntary?

DR. WILDER PENFIELD: In the eye it comes on involuntarily. Most people have had blepharal spasms. At least, I have. Such a spasm spreads to other parts of the seventh nerve and does not always stop there. It may go on to other nerves.

Two Cases of Head Injury with Damage to the Inner Ear. DR. E. A. STUART.

CASE 1.—On Sept. 22, 1939, J. B., a man aged 21, was admitted to the Montreal Neurological Institute twenty-four hours after a serious automobile accident, in which he suffered from multiple fractures of the skull, lacerations and contusions of the scalp, cerebral concussion, separation of the symphysis pubis, fracture of a transverse process of the lumbar spine and multiple fractures of the right scapula and ribs. On admission he was semicomatose. He had abrasions on the right side of the face, in the right temporal region and in the right frontal and parietal areas; edema of the right side of the scalp; a large hematoma of the right parietal region; lacerations in the right frontal region, which had been previously sutured; marked ecchymosis about the right eye; stiffness of the neck, and limitation of movement of the head and neck.

A roentgenogram of the skull made on September 22 showed multiple fractures associated with only slight depression and possibly involving several of the sinuses. Roentgenograms of the sinuses made on October 3 showed multiple fractures involving the right maxilla and zygoma. Roentgenograms of the mastoids made on October 27 showed a fracture line extending through the left petrous bone.

On admission of the patient to the hospital, the depressed fracture in the region of the right temple was elevated, débridement of the overlying lacerations was done and the wound was drained by Dr. Penfield. A fracture was found which entered the right frontal sinus and tore the dura. A small stone was removed from beneath the temporal muscle. Prior to the patient's accident he had been well, with the exception of a common cold and chronic sinal disease, localized particularly to the maxillary sinuses. On his admission to the hospital the ears were free of discharge. The drums appeared normal. There was no bleeding. There was some bleeding from the nose after the accident, but no cerebrospinal fluid leak that could be determined. Bloody discharge was present in the nasopharynx, and the right tonsil was pushed forward by a hematoma in the lateral wall of the right side of the pharynx.

On October 2 the patient complained of pain in his ears. On examination, the right middle ear was filled with blood. There was no evidence of inflammatory reaction. A whispered voice was heard from several feet. The left middle ear was filled with blood. There was a faint tinge of redness in the anterior quadrant of the drum. A whisper was heard at several feet.

On October 3 pain in the left ear was more severe and a whisper could not be heard. In the right ear a whisper was heard the full length of the room. The left drum membrane was full and bulging, and a paracentesis was done. Old blood was seen in the right middle ear. From the date of admission the patient received sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine). The left ear discharged for eight days; then the discharge ceased, and the drum membranes healed and regained a normal appearance in a short time. It was not possible to have an audiogram made until October 25. Then, on masking the right ear, it was found that there was no hearing in the left ear. At the same time there was no response from caloric tests of the left labyrinth with 10 cc. of ice water. Three days later the same observations were made in regard to the audiogram, but the left labyrinth was found to react to the caloric test.

At that time Dr. W. J. McNally expressed the opinion that both labyrinths were reacting normally and that there was a high grade nerve deafness on the left side, with no serviceable hearing in the left ear. The hearing in the right ear was within normal limits. Tinnitus was present in the left ear and persisted.

A review of the roentgenograms of the skull showed a fracture through the left internal auditory meatus, which may have involved the left cochlea. The patient was discharged from the hospital on October 28. He was able to walk, and with the exception of deafness in his left ear had made a satisfactory convalescence.

For several weeks after his discharge from the hospital the left eustachian tube was catheterized, with further audiograms being taken, but hearing has not returned in the left ear.

Cultures of the discharge from the left ear in October did not show any organisms other than Staphylococcus pyogenes, diphtheroids and Streptococcus viridans.

The Wassermann reaction of the blood was negative.

CASE 2.—On May 30, 1940, R. K., a girl aged 9 years, was admitted to the Montreal Neurological Institute because of a fractured skull which she had suffered ten days before. At the time of injury there was profuse bleeding from the right ear for two days. The patient was unconscious for nine hours and shortly after the accident was given sulfapyridine. On the ninth day after the accident her temperature became elevated and stiffness of the neck developed. She was referred to the institute as a patient with meningitis. On her admission to the hospital there was no external tenderness about the right ear. The right tympanic membrane was dull and discolored. The right ear drum was intact. The left ear drum was normal. The child appeared to be completely deaf in the right ear, with good hearing in the left ear. There was spontaneous nystagmus to the left. Roentgenograms of the skull showed a fracture passing through the right petrous bone. On account of an elevation in the cerebrospinal fluid pressure, the presence of from 25 to 40 cells in the cerebrospinal fluid and stiffness of the neck, it was considered wise to do a simple mastoidectomy on the right side. On June 1 this operation was performed.

No breaking down of the mastoid bone was observed. Blood serum was widely distributed through the cells. Some granulation tissue was found in the aditus and antrum, and a bead of pus appeared in the antrum, as though it were aspirated from the middle ear. The dura of the middle fossa and the lateral sinus were exposed and appeared healthy. Prior to the operation only a loud voice could be heard in the right ear when the left ear was masked by a Bárány noise box. After the operation the temperature promptly became normal, and no further symptoms suggestive of meningitis occurred. At the time of operation culture of material from the mastoid gave a growth of Streptococcus haemolyticus beta, group A. The Wassermann reaction of the blood was normal. The patient was discharged from the hospital on June 22. On that date rough hearing tests showed complete deafness in the right ear when the left ear was masked with a Bárány noise box. When a test was made with ice water, vestibular reactions from the right labyrinth were absent. An audiogram of the right ear without masking of the left ear showed 20 decibels of hearing over the range. However, when this was repeated with masking of the left ear, no hearing was found in the right ear. The labyrinth on the right side did not react to caloric stimulation. In two months since the patient's discharge from the hospital no improvement has been observed.

DISCUSSION

DR. J. K. M. DICKIE: The otologist is generally called in to see a head injury after the patient has nearly recovered. Physicians in Montreal are probably more fortunate than those in other places, in having the Montreal Neurological Institute to call them in on interesting cases. In regard to loss of function, meningitis

develops in a great many patients after fracture of the skull. These are usually transverse fractures, going across the labyrinth, rather than longitudinal ones. Danger of infection is of course immediate and lasts for a considerable time. With a longitudinal fracture, there is probably no great danger after the first week or so, when the tear in the middle ear is healing. There are not many cases of this type in which meningitis develops late. Meningitis associated with a transverse fracture has frequently been recorded. In 1929 Nager recorded a case in which meningitis occurred about one hundred and ninety days after fracture. The danger of subsequent meningitis is really lifelong. It has been known for thirty years or more that fractures through the petrous bone and through the labyrinth do not heal properly. They give a slight fibrous union. A case has been reported in which the patient died of meningitis sixteen years after fracture. Even though the patient recovers at the time, throughout his life he is in danger of meningitis. Nowadays sulfapyridine is given at once. When microscopic examination is done hemorrhage in the labyrinth is generally found. Small hemorrhages are also seen in the fallopian canal. In the last war it was shown that of persons who sustained injuries to the mastoid, 85 per cent had deafness afterward. Sir John Fraser described some cases of concussion from air blasts during the war, in which patients died from wounds in other parts of the body, and on microscopic examination it was found that hemorrhage into the labyrinthine cavity had occurred. I think it is easy to see why deafness, perhaps total, may follow an injury to the head even though there is no direct injury to the labyrinthine structures. With regard to the static labyrinth, most of the dizziness and other symptoms which can be attributed to injury of the vestibular structures may be due also to cerebral injury. There is usually partial return of function. Brünings described return of function two and a half years after injury. In these cases of Dr. Stuart's there are some interesting points. In the first case it is rather curious that the ears were apparently normal at first. There was no blood at first, but ten days later both ears were filled with blood. That bothered me a great deal, so I looked up the literature and found that hemorrhage into the ears after injury is a common thing. When first examined, the patient had no caloric reaction, but a short time later caloric reaction returned. There may have been some mistake in the observations, but it is reasonable to think that the patient had a hemorrhage, which later on became absorbed and allowed return of function. In the second case, meningitis came on nine days after a fracture, which seems a rather long interval. I found that a number of cases are recorded in which the cochlear function was destroyed and the vestibular function returned. It is obvious that the second patient survived only on account of surgical intervention and free drainage. It would be interesting to know whether surgical intervention and the uncovering of the area where the fracture was had any effect on the later healing of the bone. The disturbance might help to heal the bone.

Dr. G. E. Hodge: Some years ago a youth sustained an injury to the skull while playing football. No skull fracture could be found at the time. Three years later a frontal sinusitis occurred and, within a day or so, meningitis developed, and he died. I wonder what Dr. Dickie would say about that.

Dr. J. K. M. Dickie: I remember encountering a similar case. Sixteen years after a fracture meningitis developed and the patient died.

Dr. W. J. McNally: During Dr. Stuart's absence I saw the patient whose case he has reported. When I saw him, he had blood in his ears. I tested him in the room and recorded that a whisper was heard several feet from either ear. The following day the damage to both ears had progressed and paracenteses were done. When the patient was out of danger, a complete hearing test was done. He had no hearing in the left ear. The point is that he probably was deaf in the left ear at the time I tested him, but I did not use a Bárány noise box to mask the right ear.

DR. A. R. ELVIDGE: Dr. Cone observed a case in which meningitis came on a year after a fracture, and I know of one in which it came on two months after an apparently complete recovery from the effects of an injury. This infection extended upward through the nasopharynx. Sulfapyridine is used in the treatment of a good many basal skull fractures in which there are leaks, and I know of cases in which meningitis developed in spite of this. When administration of sulfapyridine was commenced, the therapeutic dose was light. However, it cured the meningitis, though it did not prevent the development of this disease in the period when conditions were acute. Often the drum membrane does not show its bluish cast until the next, or even the second, day after the accident. This discoloration is considered due to the extravasation of blood trickling down behind the membrane.

DR. KEITH HUTCHISON: Years ago the performance of a mastoidectomy in the presence of a fracture of the skull was considered dangerous and risky. Dr. Cone once invited me to see a patient who had blood in the ear two weeks after an injury. I told him it wasn't fair to ask an otologist to give evidence in a witness box when he had not seen the patient sooner than that, and he said the reason he had not called me was that he didn't want me to do any harm.

Foreign Body in the Lung. DR. H. E. MCHUGH.

D. W., a girl aged 18 months, was admitted to the hospital on Aug. 31, 1940, with grunting respiration, listlessness, anorexia and general malaise of twenty-four hours' duration. The temperature was 101 F. She was perfectly well until the evening of August 30, when listlessness and grunting respiration suddenly developed. She slept poorly that night and on the morning of admission refused to eat. No history of inhalation of a foreign body was given. Physical examination revealed nothing abnormal except dulness on percussion on the right side posteriorly, below the angle of the scapula and in the right axilla. Over the same area breath sounds were diminished. There were no rales. A tentative diagnosis of pneumonia involving the lower and middle lobes of the right lung was made.

Sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) was given on admission. On September 1 the patient's condition appeared improved. Administration of sulfapyridine was discontinued. On September 2 roentgenograms of the chest revealed a carpet tack in the right main bronchus, with collapse of the middle and lower lobes of the right lung. The trachea was displaced to the right. There was no gross displacement of the heart.

A 4 mm. bronchoscope was introduced into the trachea through an infant-sized laryngoscope and passed with some difficulty (because of the displacement of the trachea and the narrowness of the lumen) into the right main bronchus, where there was a collection of thick blood-stained mucous secretion. This was aspirated. An attempt was made to displace the point and remove the tack as quickly as possible. The first two attempts were unsuccessful. The child became cyanosed, and the bronchoscope was withdrawn and after a short rest was reintroduced. The tack was then grasped at the base of the point, but it would not enter the bronchoscope. The bronchoscope and the forceps grasping the tack were removed at the same time. Immediately after this operation it was noticed that a large collection of air was present in the subcutaneous tissues of the neck, limited above by the superficial facial attachments to the mandible. The patient was returned to the ward and placed in an oxygen tent with an ice collar about her neck.

Administration of sulfapyridine was resumed. The child breathed freely through her nose and seemed to be in no great distress. Approximately three hours later, however, she became extremely restless and cried. The result of this was a rapid extension of the emphysema over the abdomen and back. There was no emphysema beyond the inguinal ligaments. On September 3 her breathing

was somewhat more difficult. There was some indrawing of the lower end of the sternum with each inspiration, but she continued to breathe through her nose, with her mouth tightly shut. There was slight cyanosis. The respirations were noisy, and she assumed voluntarily a position of opisthotonos in the oxygen tent. The temperature was approximately 101 F., the pulse rate 130 to 160 and the respirations 32 to 40. Her condition remained about the same throughout the day, and at 7 p. m. her respiratory distress suddenly increased, cyanosis was more marked and the lower sternal and supraclavicular indrawing was more marked. In view of the patient's condition, the larynx was exposed with the Jackson infant laryngoscope. The vocal cords were perfectly normal in appearance. A 4 mm. bronchoscope would not pass freely, so an aspirator was passed blindly into the right main bronchus, and a moderate amount of thick mucous secretion was aspirated. The child's condition immediately improved, and she was returned to the oxygen tent. The condition gradually improved until the evening of September 7, when again she suddenly became cyanosed; respirations were labored and noisy. There was indrawing of the lower end of the sternum. The child appeared acutely ill. Physical examination revealed collapse of the lower lobe of the right lung. A small amount of thick mucoid secretion was aspirated through a 3 mm. bronchoscope.

On September 9 the child's condition appeared to be satisfactory. However, a roentgenogram of the chest revealed a pneumothorax, with complete collapse of the right lung but with no displacement of the trachea or of the contents of the left side of the chest. The emphysema was gradually disappearing. The child appeared to be perfectly well. The respirations varied from 24 to 36 but were not labored or noisy.

On September 19 a roentgenogram of the chest revealed that the pneumothorax on the right was disappearing. There was complete absence of emphysema.

DISCUSSION

DR. H. E. MCHUGH: This case is being reported for several reasons. The child was admitted with a short history. Prior to the onset of this illness she was perfectly well. Late at night, with no history of any possibility of inhalation of a foreign body, she suddenly acquired a respiratory stridor and noisy breathing and slept poorly. The next morning she appeared to be acutely ill. She refused to eat, and her parents brought her to the hospital. Results of the examination made at that time are shown in the notes. No roentgenograms were taken until she was seen by the attending physician. To review rapidly, the condition was at first wrongly diagnosed as pneumonia on the ground of the findings.

(Lantern slides of roentgenograms illustrating the changes in the chest were shown.)

The course was unusual. The point of exit of the air must have been the point from which I removed the tack, and the pneumothorax must be explained as the result of the bursting of a bulla following a coughing spell. The child was given sulfapyridine for ten days. No complications developed from the use of the drug. In the last two years I have encountered 3 cases such as this. One patient died, and at autopsy no pathologic condition of the larynx was observed. She had severe tracheobronchitis. I feel that in cases such as these it is only just and right that before tracheotomy is considered, the child's larynx should be explored. If there is nothing in the larynx to indicate a tracheotomy, one should not be done.

DR. D. H. BALLON: Dr. McHugh is to be congratulated on his success in this case.

DR. G. E. HODGE: Dr. McHugh has discussed this case so thoroughly that I feel there is not much left to say. He certainly is to be congratulated on his management in this case. There are two or three minor points about foreign bodies which might be considered. In children of this age there is frequently little history of aspiration of a foreign body. I think that when the condition does

not look exactly like pneumonia, roentgen examination should be made at once. The roentgenogram may not show anything, as when a vegetable foreign body is present. Recently I had as a patient a child who had a history of falling, struggling and choking while chewing peanuts two weeks previously. There had been no trouble in the interval. Roentgen examination showed no change or foreign body of any type in the lung. The next morning I examined the patient with the bronchoscope and found a peanut. I think one is justified in doing a diagnostic bronchoscopic examination. With vegetable foreign bodies the trouble comes on usually from a few hours to a few weeks after the foreign body is taken in. With metallic foreign bodies symptoms may not become manifest for months or even years. In the case reported, it is possible that the foreign body was present for some time before the symptoms developed. The difficulty to the operator in these cases should be emphasized. The 4 mm. bronchoscope is not easy to manipulate in an 18 month old child, in the presence of pus and secretion, with possibly a little struggling going on. I think Dr. McHugh did remarkably well to get the foreign body out. Daly, in reporting the removal of a prune stone from a bronchus, said that after he removed the foreign body he put in the bronchosope again and found a tear in the bronchus. I don't know whether that was done in this case, but a tear is not required for the development of emphysema. The operator is usually so pleased to get the foreign body out that he does not put the tube down again. I think this is a mistake, because often there is a plug of secretion below the foreign body, which can be aspirated. The region can then be examined. Subcutaneous emphysema occurs frequently after tracheotomy.

Emphysema is thought to be due to a ruptured alveolus, the air coming up to the hilus of the lung and thence to the neck tissues. It is governed by the valvular action of the foreign body.

I wish to note Dr. McHugh's use of sulfapyridine. I think both the postoperative and the preoperative use of the drug was justified in this case. I should like to issue a word of warning, however. Sulfapyridine won't remove a foreign body.

DR. H. E. MCHUGH: I withdrew the point of the tack within the grasp of the forceps so that I did not injure the tissues more on the way out. I believe that I may have pushed the point of the tack through, as subsequently I noted that the child had a marked extravasation of air. There was almost a definite tract from the region of the right main bronchus up to the neck, more or less through the center of the mediastinum. I felt that was how the air got there.

Transitional Cell Carcinoma. DR. D. H. BALLON.

Mrs. B. M., aged 54 years, was admitted to the hospital on Aug. 5, 1940. Since December 1939 she had had pain in her right cheek, nasal obstruction, and watery discharge from the right eye. Two months before admission, swelling appeared in the right cheek. Examination showed polypi in the right nostril and a marked hard swelling on the right cheek, which extended from the orbit to the canine fossa. Roentgenograms of the right antrum showed some destruction in the region of the orbit. A radical antrotomy was performed. The nasoantral wall, the ethmoid region and the canine fossa on the right side were found to be involved in tumor growth. A mold containing 58 mg. of radium was then inserted into the antrum and left for forty-four hours. The patient received a total of 2,552 milligram hours. This is being supplemented by roentgen treatment.

Cylindric Cell Carcinoma of the Right Ethmoid and Antrum. DR. D. H. BALLON.

Mrs. E. J., aged 50 years, was admitted to the hospital in July 1934. She complained of nasal obstruction on the right side and pain over the right cheek and frontal sinus of two years' duration. Examination showed purulent foul sanguinous discharge from the right nostril and nasopharynx, a necrotic polypoid mass in the right nostril and right choana, a swelling over the right cheek of

three weeks' duration and acute dacryocystitis. At this time the patient refused operation. In January 1935 she was readmitted on account of profuse epistaxis and a sloughing ulcer over the right cheek. The Wassermann reaction of the blood was negative. On February 4 a Moure operation was done. A mass measuring 2 inches by 1 inch by 1 inch (5 by 2.5 by 2.5 cm.) was removed from the right nostril, antrum and ethmoid region. Fifty milligrams of radium was placed in the nasoantral cavity for eighty hours. The external wound was left open. There was no evidence of recurrence until August 1940, when a swelling appeared on the hard palate, evidently a metastasis. A specimen was taken for biopsy. Carcinoma solidum was reported from the histologic examination.

Acute Suppurative Otitis Media with Meningitis Following Fracture of the Skull. DR. KEITH HUTCHISON and DR. WIGHT N. CHAPIN.

J. D., aged 7 years, was admitted to the hospital on May 25, 1940. On the morning of that date the child fell from a coal truck and struck his head on the road. He was stunned for a moment and collapsed on attempting to rise. There was no bleeding from any orifice and no convulsions. When seen in the surgical outdoor department he was semiconscious but could be roused and spoke clearly. He seemed well oriented. He vomited for the first time. On admission, the temperature was 96.5 F., the pulse rate 65 and respirations 20. On examination the child denied that he had a headache but said that he felt dizzy. He vomited again. There was a tender region in the left occipital area and an overlying hematoma of the scalp. There was no tenderness or swelling over the mastoid processes. The drum membrane of the right ear was normal. The drum membrane of the left ear was bulging, with a bluish discoloration behind it, suggestive of a hematoma of the middle ear. A small amount of old blood was present in the nasopharynx. There was spontaneous nystagmus to the right. Roentgenograms of the mastoids showed evidence of fracture extending through the left mastoid area to the middle fossa of the skull. This fracture involved the occipital bone. It ran through the petrous portion of the left temporal bone. The child's condition remained static until the fourth day of admission, when he began to complain of headache on the left side and was drowsy and irritable. There was stiffness of the neck, with a bilateral Kernig sign. Both fundi showed full dark tortuous veins. The margin of the left disk was hazy. The right disk was fairly distinct. Spontaneous nystagmus was still present. The temperature was elevated to 103.3 F. A lumbar puncture at this time showed an initial pressure of 400 mm. of water and cloudy fluid, containing numerous cells. A culture of the cerebrospinal fluid showed Pneumococcus type XVIII. On the fifth day the white blood cell count was 40,000. Examination of the left ear revealed an acute infective process. A paracentesis was done. On the seventh day of admission it was decided to do a simple mastoidectomy. At operation, when the lateral sinus plate was opened, a small layer of blood clot was found between the sinus plate and the sinus. The mastoidectomy wound was completely cleaned out. No free pus was found. With the onset of meningitis the following treatment was instituted: 60 grains (3.6 Gm.) of sulfapyridine sodium (the monohydrate sodium salt of 2-[paraaminobenzenesulfonamido]-pyridine) per day; 37½ grains (2.25 Gm.) of sulfapyridine given by mouth for three days. On the third day the level of sulfapyridine in the cerebrospinal fluid was at 10.55 mg. per hundred cubic centimeters, while that in the blood was at 15.41. The treatment with sulfapyridine was continued for twelve days, until a total of approximately 512 grains (30.7 Gm.) had been given. The patient received one blood transfusion of 150 cc. and had a continuous intravenous injection of 5 per cent dextrose in physiologic solution of sodium chloride for three days. He was treated also by lumbar puncture for one week (seventeen lumbar punctures in all). On the first day of illness he received 100,000 units of antipneumococcus type XVIII rat serum and on the second day of meningitis, 60,000. After May 28 no organisms were found in the cerebrospinal fluid.

Recurrence of Carcinoma of the Larynx Previously Treated by Hemilaryngectomy. Dr. G. E. Tremble.

A. B., a man aged 51 years, was admitted to the hospital on Aug. 21, 1939, complaining of hoarseness of six months' duration. Examination of the larynx showed that there was a fulness of the right false vocal cord. The arytenoid cartilage appeared normal, and the right true cord was fixed. No ulceration or bleeding was seen, and the left side appeared clear and moved over to the right fixed cord. A laryngofissure was decided on and was carried out on August 23. When the larynx was opened, the growth presented was found to be larger than had been anticipated, and the whole right side of the larynx and alar cartilage was involved. Therefore, a hemilaryngectomy was carried out. The pathologic diagnosis was squamous cell carcinoma. The postoperative course was satisfactory, and on September 23 the patient was discharged. At that time the tracheotomy tube had been removed for three days. The patient was eating well and had no difficulty in swallowing. Before he was discharged, he was started on a course of roentgen therapy, which lasted until October 12, with 3,750 roentgens (r) to the right side of the neck and 3,750 r to the left. The patient is now quite well and has a serviceable voice. A second course of roentgen therapy was given a few months later. The dose was increased to the point where there was danger to the overlying tissue. The patient worked regularly until June, when he began to notice dyspnea on the least exertion. This rapidly progressed until, because of external pressure, only a small chink was visible on indirect laryngoscopic inspection. No ulceration was seen. It was obvious that a recurrence had taken place, and tracheotomy was recommended, which was performed on June 27, 1940. Because of involvement of the glands in the neck, with distortion of the normal landmarks, the trachea was found with the greatest difficulty. Shortly after the tracheotomy tube was inserted, the patient gained 10 pounds (4.5 Kg.) and reurned to work. He found that it was difficult to carry on a conversation, as the opening in the tube had to be blocked to prevent the escape of air. A piece of rubber tubing attached to the tracheotomy tube enables him to talk and to carry on his normal work.

Posterior Occlusion of the Right Choana. Dr. E. A. Stuart.

A. P., a girl aged 8 years, was referred by her family physician because of nasal obstruction, thought to be due to an enlarged adenoid. On questioning the mother, one was not able to add much to this history. It was not known whether the child ever breathed through the right nostril. On examination the right nostril contained considerable mucopus. When this discharge was removed and cocaine was used in the nostril, it was discovered that an ordinary applicator would not pass through the nostril into the nasopharynx. A large adenoid was present in the nasopharynx, and the tonsils were large and unhealthy. The patient was admitted to the hospital for investigation. Her tonsils and adenoids were removed. During the operation it was established by means of a finger in the nasopharynx that a probe could not be passed through the right choana. Subsequent roentgenograms, taken with and without iodized poppyseed oil, were reported on as follows: "September 25—Question of developmental abnormality with the mesial plate of the pterygoid blocking the posterior naris on the right side."

Carcinoma Solidum of the Tongue with Metastases to the Floor of the Mouth and Mandible. Dr. E. A. Stuart.

J. C., a man aged 63 years, was admitted to the hospital on April 22, 1940, complaining of dysphonia, dysphagia, pain radiating from his throat to his ears, swelling of the tongue and bleeding from the mouth on one occasion. The symptoms first occurred in January 1940. On examination the patient was edentulous; a hard mass was present at the base of the tongue on the right side, extending to the lateral wall of the hypopharynx and as far as the epiglottis. The Wassermann

reaction of the blood was negative. Biopsy of a specimen from the base of the tongue showed carcinoma solidum. Roentgen treatment was recommended, and between April 30 and May 8 11,100 roentgen units were distributed over the right and the left anterior and posterior portions of the neck and over the intraoral regions. On September 9 the patient was readmitted, with a recurrence of his tumor at the original site. The lymph glands on both sides of the neck were palpable. On September 10 ten radon seeds were inserted into this mass, and the patient was discharged on September 14. During this admission a hard mass, measuring about 1 inch (2.5 cm.) in diameter, was noted in the midline in the floor of the mouth, directly behind the symphysis of the mandible. Roentgenograms showed erosion and destruction of the symphysis mentis. The patient was discharged from the hospital on September 14 and one week later returned for a further course of roentgen treatment to the floor of the mouth.

COLLEGE OF PHYSICIANS, PHILADELPHIA, SECTION ON OTOLARYNGOLOGY, AND PHILADELPHIA PEDIATRIC SOCIETY

KARL M. HOUSER, M.D., *Chairman*

WILLIAM HEWSON, M.D., *Clerk*

Joint Meeting, Feb. 20, 1941

Infections of the Middle Ear as Related to Other Respiratory Infections. DR. GEORGE S. FRAUENBERGER.

(The presentations were from the Central Institute and the National Forum for Deafness.)

Of the 2,529 patients admitted to the Inpatient Department of the Children's Hospital of Philadelphia, in 1939, 320 (13 per cent) had otitis media. Of this group 126 had acute purulent otitis media. The cases of these patients were selected for review. Cases of patients with contagious diseases were not included in the group.

The highest incidence of acute infection of the middle ear occurred from January to May, during which period 85 of the group were admitted. The majority (110 patients) were under 5 years of age, and 49 were under 2 years of age. The group included 77 boys and 49 girls, of whom 96 were white children and 30 were Negroes. Cultures of purulent discharge taken from the external auditory canal with a sterile swab showed mixed organisms, with Streptococcus haemolyticus, Pneumococcus and Staphylococcus aureus predominating.

Acute purulent otitis media was associated with the following conditions: acute nasopharyngitis (febrile convulsions) in 37 patients, lobar pneumonia in 33, bronchopneumonia in 15, acute bronchitis in 8, diarrhea in 4, acute tonsillitis in 4, conditions following tonsillectomy and adenoidectomy in 2 and miscellaneous maladies in 11. In regard to 12 patients with acute purulent otitis media, no history of any accompanying or preceding illness could be obtained.

Four deaths occurred in the group. Three patients were seriously ill on admission and died in less than twenty-four hours. In this group one had streptococcic meningitis with bilateral mastoiditis, another had Pneumococcus type V meningitis with bilateral mastoiditis and the third had Pneumococcus type XVIII meningitis. One patient with bronchopneumonia and mastoiditis died some days after admission.

All the patients were divided into two main groups: those in whom spontaneous drainage occurred and those who had myringotomy. Each of the groups was subdivided into patients receiving chemotherapy and those not receiving chemotherapy.

In the spontaneous rupture group (52 patients) 16 required mastoidectomy, while in the myringotomy group (74 patients) 8 required the operation. The incidence of mastoidectomy in the spontaneous rupture group as compared with that in the myringotomy group is mathematically significant with chi square 10.8. There is no mathematical significance in comparing patients receiving chemotherapy and those not receiving chemotherapy. It should be noted that many patients with spontaneous rupture were seen late in the course of the disease, and that few, if any, of the entire group received chemotherapy early. For this reason the mathematical significance mentioned is probably not of actual significance.

In the past ten years the incidence of otitis media in patients admitted to the Children's Hospital has varied between 10 and 13 per cent of the total number admitted. Similarly, during this period the incidence of mastoiditis has not shown significant change. The incidence of otitis media is unchanged since the availability of chemotherapy. Controlled studies are needed to show whether chemotherapy if given early in the course of the disease will reduce this incidence.

Relation of Infection in the Middle Ear to Deafness. DR. JAMES A. BABBITT.

A study of the structural background of infection, including natural and acquired defense barriers, avenues of infective approach and the accommodation of etiologic factors to the intricacies of the aural region, is presented. The varied types of deafness due to infection and the response of each type to the infective barrage are considered. The mechanical arrangement of the eustachian tube, tympanic cavity and mastoid is described, with emphasis on three important areas of infective exposure, namely, the anterior wall of the tympanic cavity, its posterior wall and the mesial surface. Particular attention is directed to the mesial surface, presenting the infundibulum over the lowest turn of the cochlea, and the relation of the neighboring structures to underlying paths of neurologic importance.

The developmental factors considered include conditions in infancy which render the ear more susceptible to infection and embryonic differentiation in the middle ear structure which renders certain areas more sensitive to vitamin and endocrine deficiency. Auditory imbalance, degeneration of the stria vascularis, hair cells and spiral ganglions and even demyelination of the cochlear nerve may be dependent on vitamins A, B and D.

The order of frequency of infective approach is listed as: (1) by the eustachian tube, (2) by the blood and lymph streams and (3) by the external auricular canal.

In an effort to clarify the conception of otosclerosis, an analysis of deafness, including a classification of pure conductive deafness and otosclerosis, is presented. On the basis of this perspective of infection and deafness, a correlation is suggested, with four critical phases of activity as a working basis, namely:

1. The management of acute types of middle ear involvement.

2. The contribution of the nasopharynx and adjacent portions of the eustachian tube in producing deafness.

3. The contribution of infantile otitis media toward changing the pneumatic mastoid.

4. Chronic middle ear infection and the alterations due to it in the tympanic area.

The question of diagnosis and treatment of each phase of deafness, including administration of drugs and vitamins, roentgenotherapy, tonsillectomy and other surgical procedures, is discussed.

In conclusion, the following points are stressed: (1) the frequent marvelous results obtained in progressive deafness of young children by the removal of adenoids; (2) the surprising relief frequently obtained by the correction of conditions favoring infection even in the absence of obvious infection; (3) the diminishing significance of internal ear deafness, and (4) the prophylactic significance of ordinary child hygiene, food regulation, better housing and the elimination of city noises.

DISCUSSION

DR. HORACE JAMES WILLIAMS: Dr. Babbitt has given an interesting talk. This subject deserves considerable thought.

Recently the public schools have paid increased attention to pupils with impairment of hearing, and a realization of the seriousness of a loss of hearing in so many children of school age has come about. More and more attention is directed to the recognition, diagnosis and treatment of the aural condition of these children.

Much of the deafness recognized later in life has its origin in childhood. Young children and those of school age have a greater susceptibility to infection of the upper respiratory tract than do adults. This accounts for the frequency of otitis media and mastoiditis in children.

Children who constantly have congestion of the nasal mucosa also have inflammation of the mucous membrane in the nasopharynx, including the orifice of the eustachian tube. This pathologic process extends up the eustachian tube and into the middle ear. A gradual change in the mucous membrane in the middle ear may be a forerunner of future deafness.

Adenoids around the orifice of the tube add to the congestion and should be removed when they impinge on the orifice. Their removal is not always satisfactory, because of the difficulty of inspection of the tube at operation.

When there is any evidence of impaired hearing and there is hypertrophied lymphoid tissue around the orifice of the eustachian tube, the area should be treated with radon, according to Dr. Crowe's method. This method is also of benefit in early adult life if the damage to the tube and the tympanic membrane has not been too great. Recently I have seen 2 patients treated in this way with good results.

In some patients acute suppurative otitis media develops with marked changes in the mucous membrane of the middle ear. Of these patients, some have an impairment of hearing due to interference with the passage of the sound waves through the ossicular chain. When pus remains around the oval and round windows, some of the toxins may filter through the membrane and produce a toxic effect on the organ of Corti. When this condition persists for some time, severe damage occurs, from which the organ does not completely recover.

Prophylaxis against infection in the middle ear and the early institution of proper treatment if infection does occur stand out as the best means of preventing loss of hearing.

Infants with a profuse nasal discharge should be placed on their stomachs at frequent intervals, to help keep the secretion away from the orifice of the eustachian tube. This secretion should be removed from the nose. A medicine dropper with a round tip can be used by the mother with much success.

As has been emphasized by Dr. Babbitt, proper diet, with sufficient vitamins, will do much to decrease the susceptibility to colds. A patient with a nasal infection should remain in bed in a warm well ventilated room where there is sufficient moisture.

In suppurative lesions of the middle ear the early institution of chemotherapy may prove of great value. During the last few years there has been a noticeable decrease in the number of middle ear infections from which mastoiditis developed requiring surgical intervention.

The patient with a suppurative lesion of the middle ear should remain in bed during the course of the disease. The nose and nasopharynx should be cleared of secretion. The external auditory canal should be cleaned frequently, to insure free drainage from the middle ear. When an ear has continued to discharge for two or three weeks with no tendency to subside and there are associated progressive deafness and other definite signs of mastoiditis requiring surgical treatment, an operation should be performed before there is permanent danger to the internal ear.

Book Reviews

Surgical Anatomy of the Head and Neck. By John Finch Barnhill, M.D., F.A.C.S., LL.D., and William J. Mellinger, M.D., with an introduction by Paul S. McKibben, Ph.D., LL.D. Second edition. Pp. 773, with 431 illustrations. Price, $15. Baltimore: Williams & Wilkins Company, 1940.

The second edition of Barnhill's book on the surgical anatomy of the head and neck retains the general format of the first edition, with a number of improvements.

Barnhill's accomplishments are so well known throughout the otolaryngologic world that he needs no introduction; indeed, any further comment on his integrity would be superfluous.

The associate author, too, has ably carried out certain minute anatomic studies and researches that have helped in no small measure to accomplish more fully Barnhill's aims.

This edition follows the arrangement of the previous one, with the division of the subject matter into chapters or "periods." With a few exceptions each chapter is devoted to a specific region, so that the important points of anatomy are thoroughly worked out, thus enabling the student to identify clearly those structures under consideration.

The surgical anatomy of the middle ear is given in one chapter, that of the mastoid in another and that of the petrous bone in another. The various sinuses are detailed, one chapter each being alloted to the maxillary sinus, the sphenoid sinus, the ethmoid cells and the frontal sinus. The brain, meninges, important nerves and ganglions, all of the fossae, the orbit, nose, mouth, pharynx, tongue, teeth, floor of the mouth, larynx and trachea, as well as the surgical treatment of these structures, are fully dealt with in the assigned chapters, all of the instruction being finely integrated.

Chapter 31 is entirely devoted to the subject of local anesthesia, with full discussion of its advantages and disadvantages, the consideration of apprehensive patients, the necessary instruments and method (infiltration and block) and clear detailed instruction for the technic of anesthetizing the various structures in the head and neck.

The extensive illustrations consist of photographs, drawings of dissections and various schemata. When structures required examination from different angles or special study was needed to correlate anatomic with clinical and surgical procedures more thoroughly, additional specimens were dissected for that purpose and illustrated. This is seen particularly in the chapters devoted to the petrous pyramid, the origin, course and distribution of the cranial nerves, the nasal accessory sinuses, the larynx and the intricate nerve pathways in the nose, throat and ears.

Numerous schematic drawings show vividly the anatomic features and distribution of important ganglions and the line of incision used in the standard operations on the ear, nose, throat and other structures of the head and neck. Other diagrams add to the value of the text by depicting fine points in connection with the surgical anatomy of the head and neck, references to which are often hard to find quickly in the standard works on anatomy and surgery. Some of these represent a study in themselves and might aid in solving the problems which one is apt to encounter now and again in diagnosis or in surgical procedure.

Several improvements have been made in this edition. 1. All illustrations accompany the text matter. The reader is able to correlate the instructions

immediately and to fix them in his mind. 2. The type is more easily read than that of the first edition. 3. Bold face type is used to bring out important thought units more forcibly. The authors have gone out of their way to aid the reader, lest the latter by hasty reading of the text overlook an important point.

This volume again reflects the power and skill of Dr. Barnhill, who is to be congratulated for his great presentation to American otolaryngology.

It is earnestly recommended to otolaryngologists, graduate students in otolaryngology, those taking residencies and all who practice in the fields of ophthalmology, neurology, neurosurgery, oral surgery and the various branches of dentistry, as well as to teachers of anatomy and surgery.

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Gordon F. Harkness, 215 Main St., Davenport, Iowa.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.
Place: Atlantic City.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Frank R. Spencer, Physicians' Bldg., Boulder, Colo.
President-Elect: Dr. Ralph I. Lloyd, 14–8th Ave., Brooklyn.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: Oct. 19-23, 1941.

American Broncho-Esophagological Association

President: Dr. Gabriel Tucker, 250 S. 18th St., Philadelphia.
Secretary: Dr. Paul Holinger, 1150 N. State St., Chicago.

American Laryngological Association

President: Dr. T. E. Carmody, 227–16th St., Denver, Colo.
Secretary: Dr. Charles J. Imperatori, 108 E. 38th St., New York.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. J. Mackenzie Brown, 1136 W. 6th St., Los Angeles.
President-Elect: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Place: Ambassador Hotel, Los Angeles. Time: June 16-18, 1941.

SECTIONS:

Eastern.—Chairman: Dr. N. S. Weinberger, Robert Packer Hosp., Sayre, Pa.

Southern.—Chairman: Dr. William G. Kennon, Doctors Bldg., Nashville, Tenn.

Middle.—Chairman: Dr. Walter H. Theobald, 307 N. Michigan Ave., Chicago.

Western.—Chairman: Dr. Robert C. Martin, 384 Post St., San Francisco.

American Otological Society

President: Dr. E. M. Seydell, 107 W. Douglas Ave., Wichita, Kan.
Secretary: Dr. Isidore Friesner, 36 E. 73d St., New York.

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* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

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